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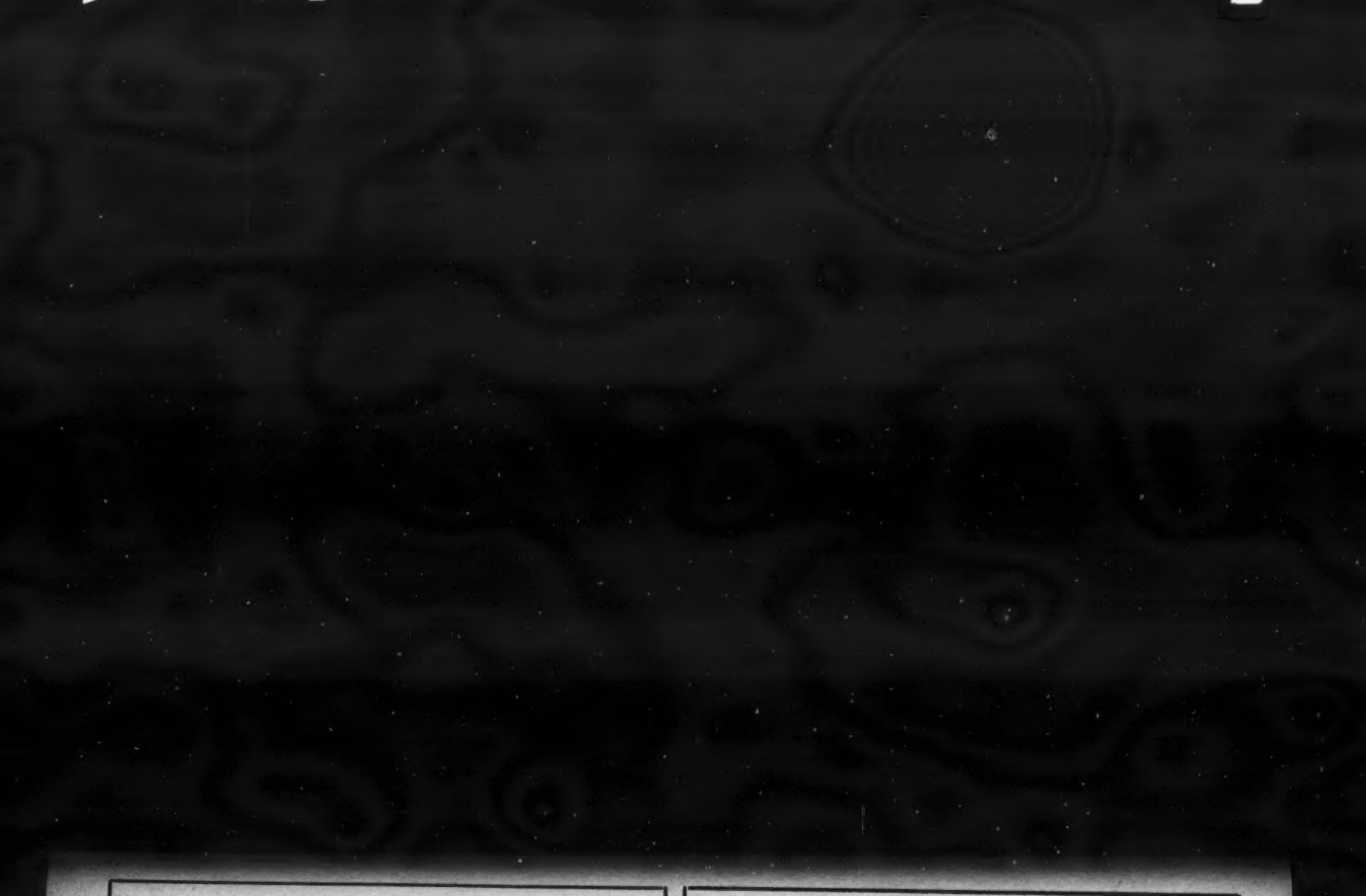

VOLUME XV SEPTEMBER, 1923 No. 3

ON THE CLASSIFICATION OF THE ASCARIDAE. III. A REVISION OF THE GENUS *DUJARDINIA* GEDOELST, WITH A DESCRIPTION OF A NEW GENUS OF ANISAKINAE FROM A CROCODILE.

By H. A. BAYLIS, M.A., D.Sc.

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(With 8 Text-figures.)

THE genus *Dujardinia* was proposed by Gedoelst (1916) for the reception of a form considered to be identical with *Ascaris helicina* Molin, 1860. In the same paper another species, *Ascaris nigra*, was described by Gedoelst, who mentions that it has close affinities with *A. helicina*, but does not actually refer it to *Dujardinia*. The present writer (1920) assigned *Ascaris halicoris* Owen, 1833, to this genus. More recent work renders it somewhat doubtful whether this species really belongs to *Dujardinia*, for reasons which will be pointed out later, and though for the present it is retained, it must be regarded as a somewhat aberrant form.

In the present paper two new species of the genus are described, and some attempt is made to revise the characters of the other species. For the opportunity of studying the type-material of the two new species, the writer is greatly indebted to Dr W. N. F. Woodland, of the Wellcome Bureau of Scientific Research.

The first question that has to be decided is the identity of the genotype of *Dujardinia*. As has been pointed out by Baylis and Daubney (1922), great confusion has arisen in the literature supposedly dealing with *Ascaris helicina*. The main points may be summarized again here. In addition to the work, already quoted, by Gedoelst, whose material came from an African crocodile, Skrjabin (1916) has published a description of what he believes to be *A. helicina*, also from an African crocodile, and proposed for the species the new genus *Trispiculascaris*. Travassos (1920), who gives no morphological grounds for his opinion, considers that neither Gedoelst's nor Skrjabin's species is really *A. helicina* Molin, and renames Gedoelst's form *Dujardinia dujardini* and Skrjabin's *Trispiculascaris trispiculascaris*.

A careful examination of African material from *Crocodilus niloticus* seems to throw some light on this question. Von Drasche (1883) re-examined and figured Molin's type-material of *Ascaris helicina*, and comparison with his

figures, especially that of the dorsal lip, shows that the material here treated as *D. helicina* almost certainly belongs to Molin's species. As pointed out by Baylis and Daubney in the paper already cited, it is also possible to reconcile Gedoelst's description with *A. helicina*, if we suppose that this author mistook the remarkable accessory piece of the male for the spicules—and this assumption, though it may seem at first sight a bold one, is borne out by the way in which his description of the spicules tallies with the actual appearance of the accessory piece in our material.

With regard to the form dealt with by Skrjabin, this author definitely states that in *Trispiculascaris* the oesophagus and intestine are simple, without blind diverticula. Unless there is an error here, therefore, it is impossible to regard the *T. helicina* of Skrjabin as a *Dujardinia*, or to regard *Dujardinia* and *Trispiculascaris* as synonymous. In any case, Skrjabin's species seems to be certainly distinct from Molin's.

As to the identity of the genotype, therefore, the writer cannot agree with the view of Travassos, and feels bound to accept the *Ascaris helicina* of Gedoelst as identical with that of Molin.

The genus is not defined by Gedoelst, except by the designation of *D. helicina* as type. The writer (1920) briefly defined it as follows:

Anisakinae: Oesophagus with a small posterior spherical bulb. An intestinal caecum present. No oesophageal appendix. Interlabia present. Dentigerous ridges absent.

Some additional characters seem to be of generic importance, and are incorporated in the following new generic diagnosis.

Dujardinia Gedoelst, 1916.

Anisakinae [Railliet and Henry, 1912, *emend.* Baylis, 1920]: Oesophagus with a small posterior spherical bulb. An intestinal caecum present. No oesophageal appendix. Interlabia present. Lips without dentigerous ridges, but with the cuticle of their internal surfaces produced into large tooth-like structures apparently capable of being interlocked. These structures are carried by three main cuticular lobes on the anterior border of each lip. Well-marked cuticular grooves run from the interlabia to the bases of the lips. Cuticle of body usually thin, with very fine and faint transverse striations. A pair of cervical papillae present at some distance behind the nerve-ring. Excretory pore at the level of the nerve-ring. Caudal end of *male* with rather well-marked lateral alae extending for a short distance in the region of the cloaca. Caudal papillae few. Two equal, slender spicules and (usually) an accessory piece of characteristic shape, with an expanded and solid head at the proximal end, and hollow and tapering distally. The lumen of the distal portion has an opening on the posterior surface of the organ. *Female* with vulva in anterior half of body, opening into a muscular, almost sucker-like "atrium" (in the genotype), from which the very long and slender vagina runs mainly in a posterior direction. The vagina ends in a small, expanded

egg-chamber, from which are given off posteriorly the two uterine branches, narrow and coiled at first, but more posteriorly expanding into two very voluminous and thin-walled tubes, filled with eggs in mature individuals and running parallel to each other. Ova with very thin, membranous shell, roundish-oval or subglobular in shape, and with contents unsegmented up to the time of laying, and usually separated by a large space from the shell.

Genotype: *D. helicina* (Molin, 1860).

The following species may be enumerated and briefly characterised. (For detailed measurements see table at p. 230.)

1. *Dujardinia helicina* (Molin, 1860).

Figs. 1 and 2.

Ascaris helicina Molin (1860), p. 337.
,, ,, v. Drasche (1883), p. 130; Pl. IX, figs. 8, 9.
,, ,, Gedoelst (1916), p. 18.
Dujardinia helicina Gedoelst (1916), p. 21.
Dujardinia dujardini Travassos (1920), p. 15.
nec Trispiculascaris helicina (Molin) Skrjabin (1916), pp. 49, 124; Pl. IV, figs. 19–21.

Hosts: *Crocodilus americanus* [*C. acutus*], (America); *C. niloticus* (Africa); *C. porosus* (India). Position: stomach.

Length, male, about 18 mm.; female, about 35–40 mm. Posterior part of body, especially in female, helicoidally twisted, invariably with a right-handed spiral. Dorsal lip (Fig. 1) with two bifurcated anterior pulp-processes projecting into the middle lobe. These processes are carried at the swollen ends of two conspicuous club-shaped masses of pulp. The bifurcated processes and the ends of the club-shaped masses are shown in the figure of the inner aspect of the dorsal lip given by v. Drasche (1883), Pl. IX, fig. 9, and it is upon this character that the identification of Molin's species mainly depends.

Accessory piece of male (Fig. 2, *a.p.*) with a median and two lateral prongs at its free end, which is slightly curved forward. Its solid proximal end is expanded and has a posterior process. Spicules very long (about 8 mm.). Caudal papillae nine pairs, of which four are postanal. The three pairs nearest to the tip of the tail small, the most posterior being lateral, the others subventral. One large pair projecting into the caudal alae just behind the cloaca and two pairs close together just in front of it. Anteriorly to these, three more large pairs at rather wide intervals. (Von Drasche appears to have been mistaken as to the position of the cloacal aperture, placing it in his figure (1883, Pl. IX, fig. 8) between the second and third instead of between the first and second of the pairs of large papillae.)

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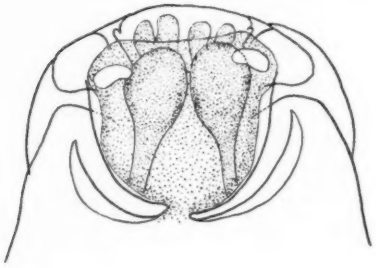

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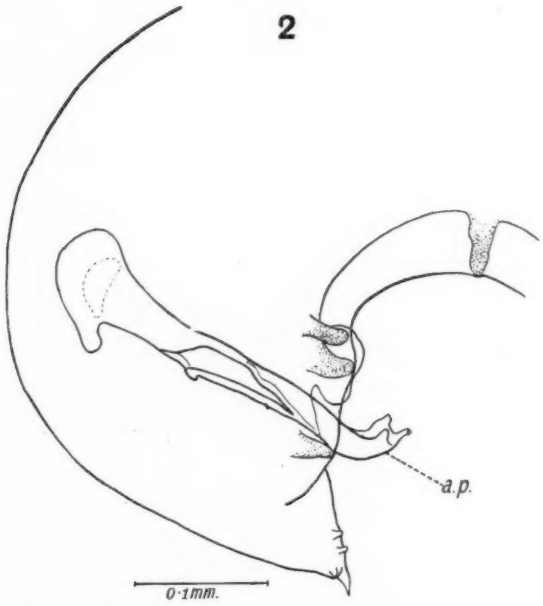


Fig. 1. *Dujardinia helicina.* Head of female; dorsal view.
Fig. 2. *Dujardinia helicina.* Caudal end of male; lateral view. *a.p.*, accessory piece.

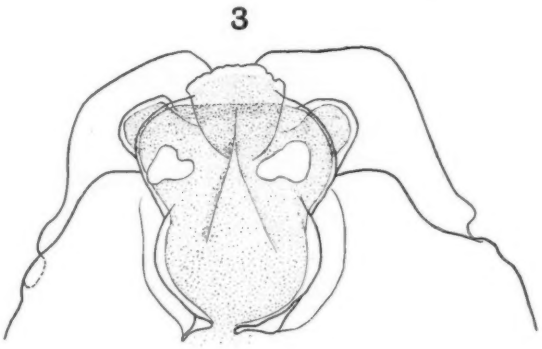
2. *Dujardinia woodlandi*, sp.n.

Figs. 3 and 4.

Host: gavial (*Gavialis gangeticus*), (India). Position: stomach.

Length, male, about 24–25 mm.; female, up to about 60 mm. Subcuticular layer consists of muscle-cells with very conspicuous, granular, mamilliform processes projecting into the body-cavity. These render the internal anatomy difficult to make out by transparency. Body apparently not twisted

in the regular helicoidal manner of *D. helicina*, but irregularly coiled. Anterior lobes of dorsal lip (Fig. 3) well-developed. No bifurcated processes seen in



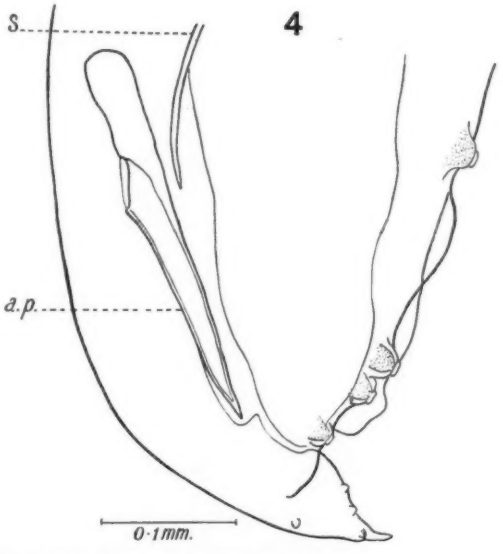


Fig. 3. *Dujardinia woodlandi*. Head of female; dorsal view.
Fig. 4. *Dujardinia woodlandi*. Caudal end of male; lateral view. *a.p.*, accessory piece; *s.*, termina portion of right spicule.

median lobe. Accessory piece of male (Fig. 4, *a.p.*) almost straight, with simple tip. Its solid proximal end not expanded, and without posterior process. Spicules short (1·25 mm.). Caudal papillae (Fig. 4) ten pairs (four postanal,

one adanal and five preanal). The four posterior pairs are small, the first and fourth being lateral, the second and third subventral.

This species is named in honour of Dr W. N. F. Woodland, who collected the material in India, in recognition of his kindness in handing it over to the writer for study. Co-types will be deposited in the British Museum (Natural History) and in the collection of the Wellcome Bureau of Scientific Research.

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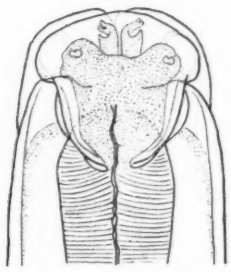

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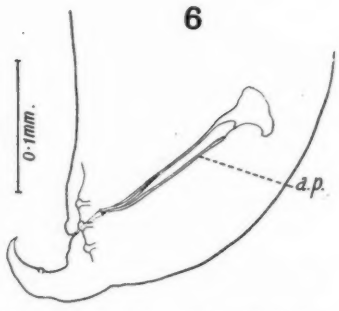

Fig. 5. *Dujardinia malapteruri.* Head of female; dorsal view.
Fig. 6. *Dujardinia malapteruri.* Caudal end of male; lateral view. *a.p.*, accessory piece.

3. *Dujardinia malapteruri*, sp.n.

Figs. 5 and 6.

Host: electric eel (*Malapterurus electricus*). Locality: Khartoum.

Length, male, about 16·5 mm.; female, up to 42·3 mm. Tail of both sexes constricted suddenly just behind the anus, and with sharply conical tip. Narrow portion of tail of male curled ventrally, that of female straight. Body shows some spiral twisting (the material, however, had been flattened out by mounting in glycerine-jelly). Dorsal lip (Fig. 5) with characteristically-shaped anterior pulp-processes, having their bifurcated terminations turned outwards

(*i.e.* laterally). Accessory piece of male (Fig. 6, *a.p.*) bent very slightly forward at the tip, which has two slender points, somewhat divergent. Its solid proximal end is expanded and has a hooked posterior process. Spicules short (1·3 mm.). Only four pairs of caudal papillae (Fig. 6) have been made out, one of which is situated on the slender portion of the tail, the others projecting into the alae at the sides of the cloaca. Probably more papillae exist, but they could not be found in the two males available.

For the opportunity of examining the type-material of this species, the writer is again indebted to Dr Woodland, the specimens having formed part of the collection of the Wellcome Bureau of Scientific Research. Co-types will be deposited there and in the British Museum (Natural History).

4. *Dujardinia halicoris* (Owen, 1833).

Ascaris halicoris Owen (1833), p. 121; Baird (1859), p. 148; Pl. LVI; Parona (1889), p. 751; figs. 1–5; Pl. XIII, figs. 1–16; v. Linstow (1906 *a*), p. 258; Pl. XI, figs. 1–10; (1906 *b*), p. 3; Pl. I, figs. 4–6.
Dujardinia halicoris Baylis (1920), p. 262.

Some additions and corrections to the existing descriptions of this species, with new figures, have been prepared for publication elsewhere[1], but its characters may be briefly summarised here.

Host: dugong (*Halicore dugong*).

Length, male, up to about 115 mm.; female, up to about 140 mm. Head separated from neck by a well-marked constriction. Well-developed interlabia present. Pulp of dorsal lip with two small conical anterior processes, converging towards the middle line. Male without accessory piece. Spicules relatively extremely short (1·1 mm.). Caudal papillae eight pairs, four postanal and four preanal, the two most anterior pairs of postanal papillae being very close together.

The absence of an accessory piece is a rather remarkable character, but in its general organisation this species seems to be very closely related to the others mentioned.

5. (?) *Dujardinia nigra* (Gedoelst, 1916).

Ascaris nigra Gedoelst (1916), p. 19.

Host: an African crocodile (probably *Crocodilus niloticus*). Position: stomach. Locality: Belgian Congo.

Length, female, 6·2–13·1 mm. From the details given in the description of this form, it seems probable that it belongs to this genus, but the specific characters mentioned are rather inadequate, and unfortunately the male is unknown.

[1] Baylis and Daubney, "A Further Report on Parasitic Nematodes in the Collection of the Zoological Survey of India," to be published by the Indian Museum. (In press.)

Table of measurements of species of Dujardinia *(in mm. unless otherwise stated).*

	helicina [Baylis]	*woodlandi* [Baylis]	*malapteruri* [Baylis]	*halicoris* [Parona, v. Linstow, Baylis]	*nigra* [Gedoelst]
Length	♂ 18 ♀ 35–40	♂ 24–25 ♀ up to 60	♂ 16·5 ♀ 42·3	♂ up to 115* ♀ up to 144*	♂ — ♀ 6·2–13·1
Thickness (maximum)	♂ 0·6 ♀ 2·2	♂ 0·6 ♀ up to 2·5	♂ 0·37–0·4 ♀ 0·9	♂ 3·16† ♀ 3·95†	♂ — ♀ 0·256–0·4
Diameter of head	♂ 0·15 ♀ 0·3	♂ 0·14 ♀ 0·3	♂ 0·11 ♀ 0·16	0·7	—
Tail, length	♂ 0·11 ♀ 0·3	♂ 0·1 ♀ 0·6–0·7	♂ 0·15 ♀ 0·55	♂ $\frac{1}{14\cdot5}$ of total length† ♀ $\frac{1}{8\cdot5}$ of total length†	♂ — ♀ $\frac{1}{4\cdot5}$ of total len
Distance from ant. end to end of oesophagus (incl. bulb)	♂ 3·4 ♀ 4·4	♂ 2·5 ♀ 3·0	♂ 2·03 ♀ 3·11	$\frac{1}{5}$–$\frac{1}{4}$ of total length†	♂♀ — ♀ $\frac{1}{5}$ of total leng
Distance from ant. end to cervical papillae	♂ 0·55 ♀ 0·8	Some distance behind nerve-ring	0·75	1·4–1·5	—
Distance from ant. end to nerve-ring	♂ 0·45 ♀ 0·65	♂ 0·5 ♀ 0·6	♂ 0·38 ♀ 0·51	1·15	$\frac{1}{3}$ of length of oesophagus
Distance from ant. end to tip of intestinal caecum	0·8	♂ 0·77 ♀ 1·1	♂ 0·66 ♀ 0·7	6·0	$\frac{4}{15}$ of length of oesophagus
Oesophageal bulb, length	♂ 0·14 ♀ 0·3	♂ 0·1 ♀ 0·15	♂ 0·12 ♀ 0·2	0·55	—
„ „ width	♂ 0·13 ♀ 0·25	♂ 0·12 ♀ 0·22	♂ 0·09 ♀ 0·15	0·6	—
Spicules, length	About 8·0	1·25	1·3	1·1	—
Accessory piece, length	0·3–0·32	0·3	0·16	[absent]	—
Vulva, distance from anterior end	About 16	About 17	17·5	About $\frac{1}{3}$ of total length	$\frac{2}{3}$ of total length
Ova, measurements	Diameter 0·075	Diameter 0·06–0·065	0·075 × 0·055	Diameter 0·13†	—

* [Parona]. † [v. Linstow].

Multicaecum, nov. gen.

The same collection from the Wellcome Bureau that contained the types of the two new species of *Dujardinia* mentioned above, included also a few preparations of an interesting Ascarid from *Crocodilus niloticus*, from the White Nile, which may with almost complete certainty be identified with *Ascaris agilis* Wedl. The structure of the alimentary canal in this form is so striking and peculiar that it seems to warrant the erection of a new genus, which has fairly close affinities with *Contracaecum* and also with *Dujardinia*, and seems, in fact, to form a link between these two genera.

The following may be given as the definition of the genus:

Anisakinae: Oesophagus with a small ventriculus from which are given off two anterior and three posterior appendices. An intestinal caecum present. Small interlabia present, with well-marked grooves running to the bases of the lips. Dentigerous ridges present. Cervical papillae not prominent, considerably behind the nerve-ring. Excretory pore slightly behind the nerve-ring. Caudal end of *male* without definite alar expansions. Two equal spicules present, and an accessory piece somewhat resembling that of *Dujardinia*. Caudal papillae few, arranged much as in *Dujardinia*. *Female* with vulva at

about the middle of the body. The muscular vagina and unpaired portion of the uterus run posteriorly. The two branches of the uterus are very short, and so narrow near the bifurcation as to contain only a single row of eggs. Eggs with thin, finely-granulated shell, of oval shape, with contents segmenting when ready for laying.

Genotype: *M. agile* (Wedl, 1862).

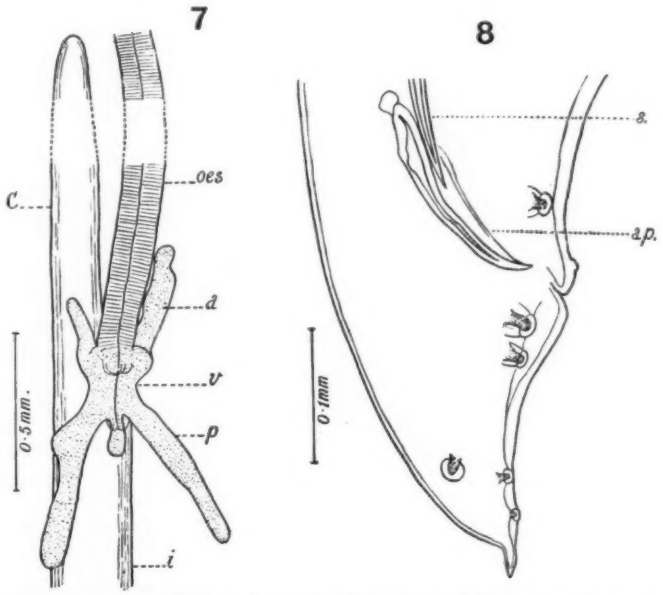

Fig. 7. *Multicaecum agile.* Portion of the alimentary canal, showing the posterior part of the oesophagus (*oes.*), the intestine (*i.*) with its caecum (*c.*), and the ventriculus (*v.*) with its anterior (*a.*) and posterior (*p.*) appendices.

Fig. 8. *Multicaecum agile.* Caudal end of male; lateral view. *a.p.*, accessory piece; *s.*, terminal portion of right spicule.

Multicaecum agile (Wedl, 1862).

Figs. 7 and 8.

Ascaris agilis Wedl (1862), p. 467; Pl. I, figs. 12–14; Pl. II, figs. 15, 16.

Host: *Crocodilus niloticus*. Position: stomach and intestine.

Length, male, 34·6 mm.; female, 31·6 mm. Maximum thickness, male, 0·65; female, 0·67 mm. (In each case the specimen was slightly flattened as the result of having been mounted in glycerine-jelly.) Diameter of head, 0·18–0·19 mm. Length of oesophagus (from anterior extremity) 4·0–4·2 mm. Cervical papillae at 1·13, nerve-ring at 0·82, excretory pore at 0·85–0·86 mm., from anterior end. Intestinal caecum extends to 1·4–1·43 mm. from anterior end. Length of oesophageal appendices: anterior, 0·2–0·34 mm.; two outer

posterior, 0·42–0·6 mm.; middle posterior, 0·15–0·22 mm. Cuticular striations 4–5 μ apart.

Tail of *male* 0·22 mm. long, gently curved ventrally, conically tapering, with suddenly narrower tip. Spicules 1·2 mm. long, rather sharply bent near their roots, which are thickened. Accessory piece about 0·2 mm. long, with simple tip and a small solid knob at the proximal end. Caudal papillae ten pairs, five pairs being postanal (of which the first, second, fourth and fifth are subventral, the third lateral) and five preanal. Of the preanal series the first, third and fourth are fairly close together and subventral; the second is large and lateral. The fifth is separated by a considerable interval from the fourth. There is also apparently a median papilla on the anterior lip of the cloaca, but this has not been definitely established, as the male specimen could only be examined in lateral view.

Tail of *female* 0·32 mm. long, straight, sharply conical, with suddenly narrower tip. A pair of caudal papillae at 0·05 mm. from the extremity. Vulva at 15·2 mm. from the anterior end of the body. Ova measure about 0·12 × 0·085 mm.

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NOTE ON THE LIFE HISTORY OF *HEMIURUS COMMUNIS* ODHNER.

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(With 2 Text-figures.)

In May 1921 and again in February 1923 a specimen of the copepod *Acartia clausi* Giesbrecht, from which was emerging a parasitic trematode, occurred in the townets. The first, taken between Rame Head and the Mewstone, outside Plymouth Sound, was a full grown female *Acartia*, nearly dead, which had the skin broken between the first and second segments of the metasome where the trematode was emerging. The second, taken close to the shore from near the mouth of the Cattewater, was a full grown male which was alive but the skin was broken between the cephalosome and metasome where the trematode was emerging in a similar way to the first.

It was evident that the worm was a young *Hemiurus*, and this is interesting because very little is known of the life-history of these trematodes and Pratt (1898) describes one from America which is also emerging from a copepod (presumably an *Acartia* or something closely related to it) in an exactly similar way to the present species. Although his worm has not been traced to its final host it was thought by Odhner (1905) to be very likely the young of *Hemiurus communis* although the vesicula seminalis has only one lobe instead of the two present in that species. Pratt states that the worms were also obtained free in the plankton as well as inside the copepods where they lived in the body cavity feeding on the blood and emerging when they had reached a certain size.

So far only these two specimens have been found at Plymouth but from them enough was seen to show that they almost certainly belong to the species *H. communis* Odhner, which is the commonest species of *Hemiurus* in this region. Like Pratt's specimens they are in a stage intermediate between the cercaria and adult, having the male organs fully developed, the female organs still incomplete. Both specimens were almost exactly the same size, when fairly well expanded being 0·64 and 0·63 mm. long, but capable of much greater elongation and retraction.

The body (Figs. 1 and 2) is narrow and more or less bluntly pointed at each end, being covered from the extreme anterior end to near the hind end with very distinct and conspicuous ridges which are so characteristic of

Hemiurus. At the hind end the "abdomen" (see Looss 1907) is invaginated and into it opens the excretory bladder. This end was in both cases the first to emerge from the copepod, the fore end coming last of all. In these young forms the "abdomen" seems to be incapable of being wholly everted. The oral sucker, placed ventrally near the anterior end measures 56 μ across, the ventral sucker, which is almost exactly twice the diameter of the oral sucker is much nearer the fore end of the body than the hind end, but varies with the contraction of the body. The mouth leads to a strong pharynx, and this to an oesophagus bifurcating about midway between the pharynx and ventral sucker into two long lobes reaching to near the cavity formed by the invagination of the "abdomen." The alimentary canal contains a yellowish food substance. The main excretory system is filled with refractive granules always

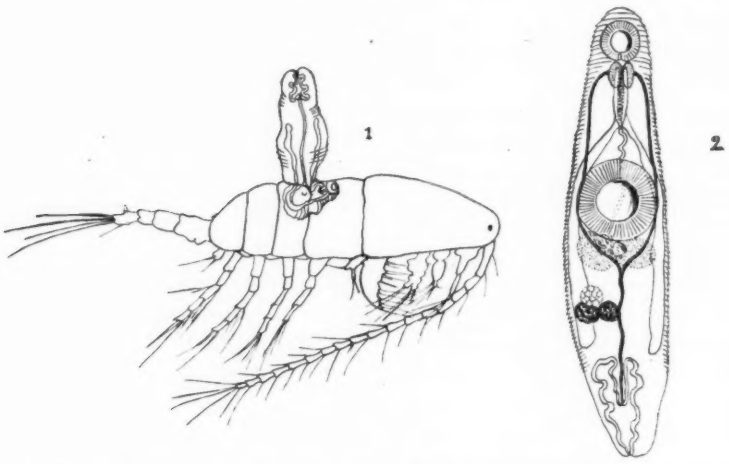

Fig. 1. *Acartia clausi* (1·7 mm. long) with *Hemiurus communis* escaping from it.
Fig. 2. *Hemiurus communis* 0·63 mm. long after escaping from *Acartia clausi*.

in movement; the fork is situated a short way behind the ventral sucker. Just behind the ventral sucker are the two testes, either on the same level or one slightly behind the other; ducts run from these to the vesicula seminalis which is composed of two thin-walled lobes immediately behind the ventral sucker from which the male duct runs forward to open at the genital pore just in front of the level of the middle of the pharynx ventrally. A female duct could not be made out, but there is a distinct round ovary behind the right testis and two small roundish vitellaria, one touching the ovary behind it and one to the left on the same level as its neighbour. In all these characters it agrees with *Hemiurus communis* and there seems no doubt that we have the intermediate host in *Acartia clausi*. Nicoll (1914) records the adult trematode in sixteen different species of fish from the English Channel:

Sparus centrodontus (= *Pagellus centrodontus*), *Capros aper*, *Lophius piscatorius*, *Cottus bubalis*, *Trigla pini*, *T. gurnardus*, *Gobius paganellus*, *Lepadogaster gouani*, *Gadus luscus*, *G. merlangus*, *G. minutus*, *G. pollachius*, *Ammodytes tobianus*, *Molva molva*, *Zeugopterus punctatus* and *Nerophis aequoreus*. It is known to occur in 30 species of British marine fishes, and, as Nicoll states, "It is, with the exception of *Derogenes varicus* the most widely distributed of all British fish parasites." He notes that it prefers gadoids, and it is to be remarked that practically all these in their young stages eat small copepods. *Acartia clausi* is one of the commonest of the copepods in the plankton and one which is eaten much by the fishes. Of those mentioned in the above list *Gobius paganellus*, *Ammodytes tobianus*, *Zeugopterus punctatus*, *Gadus luscus* and *Gadus merlangus* are known to eat *Acartia* (Lebour 1918, 19, 20), and any of the others almost certainly do when young, and many of them when adult. It is of quite frequent occurrence to find parasitic trematodes in the post-larval fishes of very small size, well under an inch. It is therefore not at all surprising that the intermediate host of one of our commonest fish trematodes should be one of our commonest copepods, especially as Pratt has already found a trematode very similar, if not identical with this one, in a copepod which is extremely like *Acartia* in America. It is probable that the eggs which escape from the fish into the sea hatch out into Miracidia which gain access to the copepod. In the body cavity of the copepod the worm feeds and grows, emerging at a certain size. It is likely that the fish eats the copepod containing the trematode which then develops in the fish, but it also may happen that the worm is eaten direct from the water just as it has escaped from the copepod. It is also conceivable that there is another host previous to the copepod in which the Miracidium may develop into a sporocyst, and a redia stage may be present, in which case the copepod would be the second intermediate host. All that we know so far is that a young stage of *Hemiurus communis* lives in the body cavity of *Acartia clausi* which acts as an intermediate host, and that several different fishes may be the final hosts. We hope for a further opportunity of studying these interesting parasites.

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A REFERENCE LIST OF THE TREMATODE PARASITES OF BRITISH MAMMALS.

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The economic importance of Trematode infections of mammals has been, and is still, felt even in this country. Epidemic liver-rot in cattle and sheep is probably one of the most serious epidemic diseases with which veterinarians have had to deal and its ravages have, on occasion, been severe. Bilharziasis in cattle is possibly of considerably less importance and, so far as I am aware, has never become endemic in this country. As pathogenic agents the Amphistomes are of third-rate importance.

The other Trematode infections of British mammals are, it may be, only of more or less academic interest, but their bearing on or relation to various zoological matters gives them an interest common to all parasitic infections.

The British mammalian fauna presents few, if any, characteristic features serving to distinguish it from that of Western Europe, and on that account one would not expect any striking differences in the parasitic fauna even in spite of the fact that the greater part of the British fauna has been separated from access to and intercourse with their congeners on the mainland for a very considerable period. It must be admitted, however, that our knowledge of the Trematode parasites of animals actually resident in Britain is extraordinarily scanty. Except in the case of the domesticated mammals the number of investigations that have been made is practically negligible.

In this instance, as in so many others, we are indebted for almost all the knowledge we possess to continental investigators. The only British name of any consequence that occurs is that of Cobbold.

As in most other departments of helminthology the solid foundations of our knowledge were laid by Rudolphi (1809–1819)[1]. Thirty years later Dujardin's entertaining *Histoire des Helminthes* (1845) helped to give the subject a semi-popular interest, in France at any rate. After the lapse of another twenty years Cobbold (1855–1879) performed a similar service in this country and a large amount of interesting material appears to have passed through his hands. Von Linstow (1876–1886) added something to our knowledge of the larval Holostomes, while Poirier (1885–1886) supplied considerable

[1] The dates in brackets refer to the period during which the author dealt particularly with the Trematode parasites of Mammals.

information on the Amphistomes and the parasites of marine mammals. Brandes (1888–1898) was the first to tackle the difficult problem of Holostome structure and classification in general.

It is to Looss (1885–1907) and Braun (1892–1902), however, that we owe much of our present general knowledge of mammalian Trematode parasites, and their work has traversed the whole field of both domesticated and non-domesticated mammals. More recently the work of Stiles (1894–1910) and Fischoeder (1901–1904) has served to consolidate our knowledge of the economically important Amphistome parasites of domesticated cattle.

Amongst the most recent workers who have contributed materially to the subject are Odhner (1905–1914) and Ransom (1920).

In addition to the list of parasites of the native fauna I have added a list of those of the domesticated mammals. While the latter list contains records of many parasites which have not been met with in this country nor are likely to become indigenous it has seemed advisable to make the list as complete as possible owing to the fact that such animals are from time to time imported and their parasites may so become a matter of interest. Check lists of such parasites have already been published by Leiper (1911–1912), but as these lists are unprovided with references to literature their usefulness is considerably restricted.

Apart from *Fasciola hepatica*, the common liver-fluke, there is no particularly common or characteristic species of mammalian Trematode in Britain, with the possible exception of *Echinostomum melis* Schrank, better known as *E. trigonocephalum* Rud.

The subject of mammalian Trematodes is naturally of some interest and concern in human helminthological investigations in view of the fact that at least a dozen of the species noted in the present list have at one time or other been recorded as parasites of man.

It will be seen that there are about a dozen unclassified species, most of which are *species inquirendae* or of doubtful validity.

LIST OF THE TREMATODE PARASITES OF BRITISH MAMMALS ARRANGED SYSTEMATICALLY.

I. DISTOMATA.

I. ECHINOSTOMIDAE Dietz

i. *Echinostomum* Rudolphi 1809

1. E. spiculator (Duj.)	Dujardin 1845, v. Linstow 1886
2. E. acanthoides (Rud.)	Rudolphi 1819, Braun 1901
3. (E.) exasperatum Rud.	Rudolphi 1819, Braun 1901

ii. *Euparyphium* Dietz

4. E. melis (Schrank) [=E. trigonocephalum Rud.]	Dietz 1910, Mühling 1898

iii. *Echinochasmus* Dietz

5. E. perfoliatus (Ràtz) [=E. gregale Raill. and Hen.]	Ràtz 1908, Ciurea 1915 *a* Railliet and Henry 1909

II. Opisthorchiidae Braun

i. *Opisthorchis* R. Blanch. 1895

6. O felineus (Riv.) — Braun 1895, Barker 1911
7. O tenuicollis (Rud.) — Braun 1893 *b*, Barker 1911
8. O wardi Wharton — Wharton 1921
9. O conjunctus (Cobbold) — Cobbold 1879, Braun 1895, Stiles and Hassall 1894

ii. *Amphimerus* Barker 1911 (=*Paropisthorchis* Stephens 1912)

10. A. pseudofelineus (Ward) — Ward 1895, Barker 1911
11. A. noverca Braun [=P. caninum (Barker)] — Barker 1911, Leiper 1913

iii. *Clonorchis* Looss 1907

12. C. sinensis (Cobbold) [=C. endemicus Baelz] — Looss 1907 *c*, Kobayashi 1911

iv. *Metorchis* Looss 1899

13. M. albidus (Braun) — Braun 1893 *b*, Stiles and Hassall 1894, Ciurea 1915 *b*

v. *Parametorchis* Skrjabin 1913

14. P. complexus (St. and H.) — Stiles and Hassall 1894, Skrjabin 1913 *a*

vi. *Pseudamphistomum* Lühe 1908

15. P. truncatum (Rud.) [=D. conus Crepl. =D. campanulatum Ercolani] — Lühe 1908, Braun 1893 *b*
16. P. danubiense Ciurea — Ciurea 1913

vii. *Cyclorchis* Lühe 1908

17. C. campula (Cobbold) — Cobbold 1876, 1879, Lühe 1908

viii. *Omphalometra* Looss 1899

18. O. flexuosa (Rud.) — Mühling 1896, Looss 1899

III. Fasciolidae Railliet

a. Fasciolinae Looss

i. *Fasciola* Linn. 1758

19. F. hepatica Linn. — Sommer 1880, Thomas 1883, Blanchard 1896
20. F. gigantica Cobbold [=F. gigantea Diesing =F. angusta Raill. =F. aegyptiaca Looss] — Cobbold 1855, Blanchard 1896, Faust 1920, Jackson 1921

ii. *Fascioloides* Ward 1917

21. F. magna (Bassi) — Ward 1895, 1917

b. Fasciolopsinae Odhner

iii. *Fasciolopsis* Looss 1899

22. F. buskii (Lankester) — Odhner 1902, 1909, Ward 1909 Goddard 1919, Nakagawa 1921
23. (F.) tursionis (Marchi) — Marchi 1873

c. Brachycladiinae Odhner

iv. *Brachycladium* Looss 1899

24. B. oblongum (Braun) — Cobbold 1858, Stiles 1895, Braun 1900 *a*
25. B. palliatum (Looss) — Looss 1885, Stiles 1895
26. B. delphini (Poirier) — Poirier 1886, Stiles 1895
27. B. rochebruni (Poirier) — Poirier 1886, Stiles 1895

v. *Lecithodesmus* Braun 1902

28. L. goliath (v. Ben.) — Braun 1902, Odhner 1905

III. FASCIOLIDAE Railliet (*continued*)
vi. *Orthosplanchnus* Odhner 1905
29. O. arcticus Odhner — Odhner 1905
30. O. fraterculus Odhner — Odhner 1905

IV. ALLOCREADIIDAE Odhner
i. *Crepidostomum* Braun 1900
31. C. metoecus (Braun) — Braun 1900 *b*

V. LEPODERMATIDAE Odhner
i. *Lepoderma* Looss 1899
32. L. vespertilionis (Mueller) [=D. lima Rud.] — Braun 1900 *b*
33. L. asperus Stoss. — Stossich 1904
ii. *Opisthioglyphe* Looss 1899
34. O. loccllus Kossack — Kossack 1910

VI. DICROCOELIIDAE Odhner
i. *Dicrocoelium* (Dujardin 1845)
35. D. lanceatum Stiles and Hass. [=D. lanceolatum (Mehlis) =D. dendriticum (Rud.) Odhner] — R. Blanchard 1888, Braun 1895, Stiles and Hassall 1898, 1904
36. D. hospes Looss — Looss 1907 *b*
ii. *Eurytrema* Looss 1907
37. E. coelomaticum (Giard and Billet) — Giard and Billet 1892, Looss 1907 *c*
38. E. pancreaticum (Janson) — Katsurada and Saito 1906, de Does 1907, Looss 1907 *c*
39 E. parvum Senoo — Senoo 1907
iii. *Ityogonimus* Lühe 1899
40. I. ocreatus (Goeze) [=I. lorum (Duj.)] — Melnikov 1865, Gonder 1910
41. I. filum Looss — Looss 1907 *a*

VII. LECITHODENDRIIDAE Odhner
i. *Lecithodendrium* Looss 1896
42. L. lagena (Brandes) [=D. ascidia v. Ben. *nec* Rud.) — Brandes 1888, Looss 1899
43. L. chilostomum (Mehlis) [=D. ascidioides v. Ben.] — Linstow 1878, Braun 1900 *b*
ii. *Pycnoporus* Looss 1899
44. P. heteroporus (Duj.) — Brandes 1888, Looss 1899
45. (P.) macrolaimus (v. Linst.) — Linstow 1894, Braun 1900 *b*

VIII. HETEROPHYIDAE Odhner
i. *Heterophyes* Cobbold 1866
46. H. heterophyes (v. Siebold) — Looss 1894, 1902, Ransom 1920
47. H. fraternus (Looss) — Looss 1894, 1902, Ransom 1920
48. H. aequalis Looss — Looss 1902, Ransom, 1920
49. H. dispar Looss — Looss 1902, Ransom 1920
ii. *Ascocotyle* Looss 1899
50. A. minuta Looss — Looss 1899, Ransom 1920
51. A. italica Alessandrini [=Echinostomum piriforme Blanc and Hedin] — Alessandrini 1906, Ransom 1920
iii. *Loxotrema* Kobayashi 1912 [=*Metagonimus* Katsurada 1913 = *Yokogawa* Leiper 1913]
52. L. ovatum Kobayashi [=M. yokogawai (Katsurada)] — Kobayashi 1912, Yokogawa 1913, Ransom 1920

VIII. HETEROPHYIDAE Odhner (*continued*)

iv. *Cotylophallus* Ransom 1920

53. C. venustus Ransom — Ransom 1920
54. C. similis Ransom — Ransom 1920

v. *Centrocestus* Looss 1899

55. C. cuspidatus (Looss) — Looss 1896, Ransom 1920

vi. *Cryptocotyle* Lühe 1899

56. C. lingua (Creplin) — Jägerskiöld 1898, Ransom 1920

vii. *Galactosomum* Looss 1899

57. (G.) erinaceum (Poirier) — Poirier 1886

viii. *Parabascus* Looss 1907

58. (P.) semisquamosus (Braun) — Braun 1900 *b*, Looss 1907 *b*

ix. [*Eurysoma* Duj. 1845]

59. E. squamula (Rud.) — Rudolphi 1819, Dujardin 1845, Zeller 1867, Fraipont 1880

IX. TROGLOTREMIDAE Odhner

i. *Troglotrema* Odhner 1914

60. T. acutum (F. S. Leuckart) — Leuckart 1842, Odhner 1914

ii. *Paragonimus* Braun 1899

61. P. westermanii (Kerbert) — Katsurada 1900, Kubo 1912, Ward and Hirsch 1915

62. P. kellicotti Ward — Ward 1908, Ward and Hirsch 1915

iii. *Pholetor* Odhner 1914

63. P. gastrophilus (Kossack) — Kossack 1910, Odhner 1914

X. HARMOSTOMIDAE Odhner

i. *Harmostomum* Braun 1899

64. H. erinacei (Blanch.) — Looss 1894, Hofmann 1899
[= H. linguaeformis (Dies.)
= H. leptostomum (Olsson)
= H. caudale (v. Linstow)]

65. H. spinosulum (Hofmann) — Hofmann 1899, Braun 1901

XI. SCHISTOSOMIDAE Looss 1899 = BILHARZIIDAE Odhner 1912

i. *Schistosoma* Weinland 1858 (= *Bilharzia* Cobbold 1859)

66. S. bovis (Sonsino) — Blanchard 1895, Stiles 1898, Skrjabin 1911
[= S. crassum Sonsino]

67. S. indicum Montgomery — Montgomery 1906

68. S. japonicum Katsurada — Katsurada 1904, 1913, Kerr 1905, Looss 1905 *a*

69. S. spindalis Montgomery — Montgomery 1906, Vryburg 1907

70. S. turkestanicum Skrjabin — Skrjabin 1913 *b*

XII. UNCLASSIFIED GENERA

i. *Mesotretes* Braun 1900

71. M. peregrinus Braun — Braun 1900 *b*

ii. *Odhneriella* Skrjabin 1915

72. O. rossica Skrjabin — Skrjabin 1915

iii. *Microtrema* Kobayashi 1915

73. M. truncatum Kobayashi — Kobayashi 1915

XIII. UNCLASSIFIED SPECIES

74. Distomum advena Duj. [=D. migrans Duj.]	Dujardin 1845, Braun 1893 *a*
75. D. aristotelis Stoss.	Stossich 1892, Braun 1900 *b*
76. D. instabile Duj.	Dujardin 1845, Braun 1901
77. D. musculi Rud. [=D. recurvum Duj.]	Dujardin 1845, Braun 1901
78. D. Pallasii Poirier	Poirier 1885, Buttel-Reepen 1900
79. D. philocholum Creplin	Creplin 1845
80. D. pusillum (Braun)	Rudolphi 1809, Dujardin 1845
81. D. Putorii Molin (*nec* Schrank)	Molin 1861, Linstow 1875
82. D. soricis Diesing	Diesing 1858, Braun 1893 *a*
83. "D. truncatum" F. S. Leuckart (*nec* Rudolphi)	Leuckart 1842, Braun 1901
84. D. vitta Duj.	Dujardin 1845, Braun 1901
85. D. suis Stiles	Stiles 1898, Stiles and Hassall 1900

II. MONOSTOMATA.

i. *Ogmogaster* Jägerskiöld 1891

86. O. plicatus (Creplin)	Jägerskiöld 1891, Braun 1892

ii. *Notocotylus* Diesing 1839

87. N. noyeri Joyeux	Joyeux 1922
88 Monostomum vespertilionis Rud.	Rudolphi 1819, Braun 1893 *a*

III. HOLOSTOMATA.

i. *Alaria* Schrank 1788

89. A. alata (Goeze)	Brandes 1890, Hall and Wigdor 1918
90. A. michiganensis Hall and Wigdor	Hall and Wigdor 1918
91. A. americana Hall and Wigdor	Hall and Wigdor 1918

ii. *Pharyngostomum* Ciurea 1922

92. P. cordatum (Diesing)	Brandes 1890, Ciurea 1922

iii. *Diplostomum* v. Nordmann 1832

93. D. Putorii v. Linstow	Linstow 1877, Brandes 1890

iv. *Tetracotyle* Filippi 1854

94. T. foetorii v. Linstow	Linstow 1876, Faust 1918
95. T. soricis v. Linstow	Linstow 1877, Braun 1892

IV. AMPHISTOMATA.

PARAMPHISTOMIDAE Fischoeder

Paramphistominae Fischoeder

i. *Paramphistomum* Fischoeder 1901

96. P. cervi (Schrank)	Fischoeder 1902, 1903, Stiles 1898
97. P. calicophorum Fischoeder	Fischoeder 1902, 1903
98. P. explanatum (Creplin)	Fischoeder 1904, Stiles 1898
99. P. scoliocoelium Fischoeder	Fischoeder 1904
100. P. tuberculatum (Cobbold)	Fischoeder 1902, Stiles 1898

ii. *Cotylophoron* Stiles and Goldberger 1910

101. C. cotylophorum (Fischoeder)	Fischoeder 1901, 1903
102. C. indicum Stiles and Goldberger	Stiles and Goldberger 1910

iii. *Verodunia* Lahille and Joan 1917

103. V. tricoronata Lahille and Joan	Lahille and Joan 1917

PARAMPHISTOMIDAE Fischoeder (*continued*)
Stephanopharynginae Stiles and Goldberger
iv. *Stephanopharynx* Fischoeder 1901
104. S. compactus Fischoeder — Fischoeder 1902, 1903

Cladorchiinae Fischoeder
v. *Pseudodiscus* Sonsino 1895
105. P. collinsii (Cobbold) — Cobbold 1875, Sonsino 1895, Stiles and Goldberger 1910
106. P. stanleyi (Cobbold) — Cobbold 1875, Stiles and Goldberger 1910

GASTROTHYLACIDAE Stiles and Goldberger
i. *Gastrothylax* Poirier 1883
107. G. crumenifer (Creplin) — Brandes 1898, Fischoeder 1903
108. G. compressus Brandes — Brandes 1898, Fischoeder 1903

ii. *Fischoederius* Stiles and Goldberger 1910
109. F. cobboldii (Poirier) — Stiles 1898, Fischoeder 1903
110. F. elongatus (Poirier) — Stiles 1898, Brandes 1898, Fischoeder 1903

iii. *Carmyerius* Stiles and Goldberger 1910
111. C. spatiosus (Brandes) — Brandes 1898, Fischoeder 1903
112. C. mancupatus (Fischoeder) — Fischoeder 1902, 1903
113. C. gregarius (Looss) — Looss 1896, Brandes 1898, Stiles 1898

GASTRODISCIDAE Stiles and Goldberger
i. *Gastrodiscus* Leuckart 1877
114. G. aegyptiacus (Cobbold) — Sonsino 1895, Looss 1896
115. G. polymastos Leuckart — Leuckart 1880, Lejtenyi 1880, Otto1896
116. G. secundus Looss — Looss 1907 *c*
117. G. minor Leiper — Leiper 1913

ii. *Gastrodiscoides* Lewis and McConnell 1876
118. G. hominis Lewis and McConn. — Stephens 1906, Leiper 1913

iii. *Homalogaster* Poirier 1883
119. H. poirieri Giard and Billet — Giard and Billet 1892, Stiles and Goldberger 1908

LIST OF THE TREMATODE PARASITES OF BRITISH MAMMALS ARRANGED ACCORDING TO THEIR HOSTS.

I. RODENTIA

1. *Sciurus leucourus* Kerr. Squirrel
[=*S. vulgaris* Linn.]
Fasciola hepatica Abildg. — Liver and gall-bladder

2. *Apodemus sylvaticus* Linn. Long-tailed field mouse
Distomum musculi Rud. — Intestine
Distomum vitta Duj. — Intestine

3. *Mus musculus* Linn. House mouse
Distomum musculi Rud. — Intestine
Distomum vitta Duj. — Intestine

4. *Mus norvegicus* Erxl. Brown rat
Echinostomum spiculator Duj. — Small intestine

5. *Arvicola amphibius* Linn. Water vole
Notocotylus noyeri Joyeux — Intestine

6. *Lepus timidus* Linn. Mountain hare
Fasciola hepatica Abildg. — Liver and gall-bladder
Dicrocoelium lanceatum St. and H. — Liver, gall-bladder and intestine

I. RODENTIA (*continued*)	
7. *Oryctolagus cuniculus* (Linn.) Rabbit	
Fasciola hepatica Abildg.	Liver
Dicrocoelium lanceatum St. and H.	Liver
II. CARNIVORA	
8. *Felis sylvestris* Schreb. Wild cat	
Pharyngostomum cordatum (Dies.)	Intestine
9. *Vulpes vulpes* Linn. Fox	
Alaria alata (Dies.)	Intestine
Echinostomum melis (Schrank)	Intestine
Opisthorchis conjunctus (Cobbold)	Bile duct
Opisthorchis felineus (Rud.)	Gall-bladder
Pseudamphistomum truncatum (Rud.)	Gall-bladder
Metorchis albidus Bru.	Gall-bladder and liver
Heterophyes heterophyes (v. Sieb.)	Intestine
10. *Martes martes* Linn. Pine marten	
Echinostomum melis (Schrank)	Intestine
11. *Putorius putorius* Linn. Pole cat.	
Echinostomum melis (Schrank)	Intestine
Eurysoma squamula (Rud.)	Intestine
Troglotrema acutum (Leuckart)	Frontal and ethmoid sinuses
Distomum Putorii Molin (larva)	Thoracic cavity and muscles in cysts
Diplostomum Putorii v. Linst.	Oesophagus and intestine in cysts
Tetracotyle foetorii v. Linst.	Skin and muscles in cysts
12. *Mustela nivalis* Linn. Weasel	
Echinostomum melis (Schrank)	Intestine
13. *Meles meles* Linn. Badger	
Echinostomum melis (Schrank)	Intestine
Harmostomum erinacei (Blanch.)	Intestine
14. *Lutra lutra* Linn. Otter	
Echinostomum melis (Schrank)	Intestine
15. *Halichoerus gryphus* Fabr. Grey seal	
Opisthorchis tenuicollis (Rud.)	Liver
Metorchis albidus (Braun)	Liver
Pseudamphistomum truncatum (Rud.)	Liver
16. *Phoca vitulina* Linn. Common seal	
Echinostomum acanthoides (Rud.)	Intestine
Pseudamphistomum truncatum (Rud.)	Liver
Cotylophallus similis Ransom	Intestine
Cryptocotyle lingua (Creplin)	Intestine
17. *Phoca groenlandica* Fabr. Harp seal	
Pseudamphistomum truncatum (Rud.)	Gall-bladder
18. *Erignathus barbatus* Fabr. Bearded seal	
Opisthorchis tenuicollis (Rud.)	Liver
Orthosplanchnus arcticus Odhner	Gall-bladder
19. *Odobaenus rosmarus* Linn. Walrus	
Orthosplanchnus fraterculus Odhner	Gall-bladder
Odhneriella rossica Skrjabin	Liver
III. CETACEA	
20. *Phocaena communis* Less. Porpoise	
Brachycladium oblongum Cobbold	Bile ducts
Pholetor gastrophilus Kossack	Stomach in cysts
Distomum Pallasii Poirier	Stomach
Distomum philocholum Crepl.	Liver

III. Cetacea (*continued*)

21. *Delphinus delphis* Linn. Dolphin	
Brachycladium palliatum (Looss)	Bile ducts
Brachycladium Delphini (Poirier)	Liver
Brachycladium Rochebruni (Poirier)	Liver
(Galactosomum) erinaceum (Poirier)	Intestine in cysts
22. *Tursiops tursio* Bonn. Bottle-nosed Dolphin	
(Fasciolopsis) tursionis (Marchi)	Intestine
23. *Balaenoptera borealis* Less. Rudolphi's rorqual	
Lecithodesmus goliath (v. Ben.)	Liver

IV. Ungulata

24. *Cervus elaphus* Linn. Red deer	
Fasciola hepatica Abildg.	Gall-bladder and liver
Fascioloides magna (Bassi)	Liver
Dicrocoelium lanceatum St. and H.	Gall-bladder, liver and intestine
Paramphistomum cervi (Schrank)	Stomach
25. *Cervus dama* Linn. Fallow deer	
Fasciola hepatica Abildg.	Gall-bladder and liver
Fascioloides magna (Bassi)	Liver
Dicrocoelium lanceatum St. and H.	Gall-bladder and intestine
Paramphistomum cervi (Schrank)	Stomach
26. *Capreolus capreolus* Linn. Roe deer	
Fasciola hepatica Abildg.	Liver and gall-bladder
Paramphistomum cervi (Schrank)	Stomach

V. Insectivora

27. *Erinaceus europaeus* Linn. Hedgehog	
Echinostomum melis (Schrank)	Intestine
Harmostomum erinacei (Blanch.)	Intestine
Harmostomum spinosulum (Hofmann)	Intestine
Distomum pusillum Braun	Mesentery and connective tissue in cysts
28. *Talpa europaea* Linn. Mole	
Omphalometra flexuosa (Rud.)	Stomach and intestine
Ityogonimus ocreatus (Goeze)	Intestine
Ityogonimus filum Looss	Intestine
29. *Sorex araneus* Linn. Common shrew	
Distomum advena Duj.	Intestine
Distomum soricis Pontallié	Gall-bladder
Tetracotyle soricis v. Linst.	Intestine
30. *Neomys fodiens* Schreb. Water-shrew	
Opisthioglyphe locellus Kossack	Intestine
(Echinostomum) exasperatum Rud.	Intestine
Distomum instabile Duj.	Intestine
"Distomum truncatum" F. S. Leuck.	Kidneys

VI. Chiroptera

31. *Rhinolophus ferrum-equinum* Schreb. Greater horse-shoe bat	
Lepoderma vespertilionis (O. F. Müller)	Intestine
Mesotretes peregrinus (Braun)	Intestine
32. *Rhinolophus hipposideros* Bechst. Lesser horse-shoe bat	
Lepoderma vespertilionis (Müller)	Intestine
Lecithodendrium lagena (Brandes)	Intestine
Lecithodendrium chilostomum (Mehlis)	Intestine

VI. CHIROPTERA (*continued*)

33. *Plecotus auritus* Linn. Long-eared bat	
Lepoderma vespertilionis (Müller)	Intestine
Lepoderma asperum (Stossich)	Intestine
Lecithodendrium lagena (Brandes)	Intestine
Lecithodendrium chilostomum (Mehlis)	Intestine
34. *Vespertilio serotinus* Schreb. Serotine	
Lecithodendrium lagena (Brandes)	Intestine
Lecithodendrium chilostomum (Mehlis)	Intestine
35. *Nyctalus noctula* Schreb. Noctule	
Crepidostomum metoecus (Braun)	Intestine
Lepoderma vespertilionis (Müller)	Intestine
Lecithodendrium chilostomum (Mehlis)	Intestine
(Parabascus) semisquamosus Braun	Intestine
Distomum aristotelis Stossich	Intestine
Monostomum vespertilionis Rud.	Intestine
36. *Nyctalus leisleri* Kuhl. Leisler's bat	
Lecithodendrium chilostomum (Mehlis)	Intestine
37. *Pipistrellus pipistrellus* Schreb. Pipistrelle	
Lepoderma vespertilionis (Müller)	Intestine
Lecithodendrium chilostomum (Mehlis)	Intestine
Lecithodendrium lagena (Brandes)	Intestine
Pycnoporus heteroporus (Duj.)	Intestine
(Pycnoporus) macrolaimus v. Linst.	Intestine
38. *Leuconoe Daubentoni* (Kuhl). Daubenton's bat	
Lepoderma vespertilionis (Müller)	Intestine
Lecithodendrium lagena (Brandes)	Intestine
Lecithodendrium chilostomum (Mehlis)	Intestine
39. *Myotis nattereri* Kuhl. Natterer's bat	
Lepoderma vespertilionis (Müller)	Intestine
Lecithodendrium chilostomum (Mehlis)	Intestine
40. *Myotis mystacinus* (Leisler). Whiskered bat	
Lepoderma vespertilionis (Müller)	Intestine
Lecithodendrium lagena (Brandes)	Intestine
Lecithodendrium chilostomum (Mehlis)	Intestine

DOMESTICATED MAMMALS.

1. *Felis domestica* L. Cat	
Echinostomum melis (Schrank)	Intestine
Echinochasmus perfoliatus (v. Ràtz)	Intestine
Opisthorchis felineus (Riv.)	Gall-bladder
? Opisthorchis tenuicollis (Rud.)	Liver and gall-bladder
Opisthorchis wardi Wharton	Bile duct
Opisthorchis conjunctus (Cobbold)	Bile duct
Amphimerus pseudofelineus Ward	Gall-bladder and bile duct
Clonorchis sinensis	Liver
Metorchis albidus Braun	Liver and gall-bladder
Parametorchis complexus (St. and H.)	Gall-bladder and bile duct
Pseudamphistomum truncatum (Rud.)	Gall-bladder
Pseudamphistomum danubiense Ciurea	Liver
? Fasciola hepatica L.	Liver
Dicrocoelium lanceatum (St. and H.)	Gall-bladder
Heterophyes heterophyes (v. Siebold)	Intestine
Heterophyes fraternus Looss	Intestine
Heterophyes aequalis Looss	Intestine
Heterophyes dispar Looss	Intestine

1. *Felis domestica* L. (*continued*)

Ascocotyle minuta Looss	Intestine
Loxotrema ovatum Kobayashi	Intestine
Cotylophallus venustus Ransom	Intestine
Paragonimus westermanii (Kerb.)	Lungs
Schistosoma japonicum Katsurada	Veins
Pharyngostomum cordatum (Diesing)	Intestine

2. *Canis familiaris* Linn. Dog

? Echinostomum melis (Schrank)	Intestine
Echinochasmus perfoliatus (v. Ràtz)	Intestine
Opisthorchis felineus (Riv.)	Gall-bladder
? Opisthorchis tenuicollis (Rud.)	Liver and gall-bladder
Opisthorchis conjunctus (Cobbold)	Bile duct
Clonorchis sinensis (Cobbold)	Liver
Amphimerus noverca (Braun)	Liver
Metorchis albidus Braun	Liver and gall-bladder
Pseudamphistomum truncatum (Rud.)	Gall-bladder
Pseudamphistomum danubiense Ciurea	Liver
Dicrocoelium lanceatum (St. and H.)	Liver
Heterophyes heterophyes (v. Sieb.)	Intestine
Heterophyes fraternus Looss	Intestine
Heterophyes aequalis Looss	Intestine
Heterophyes dispar Looss	Intestine
Ascocotyle minuta Looss	Intestine
Ascocotyle italica Alessandrini	Intestine
Loxotrema ovatum Kobayashi	Intestine
Cotylophallus venustus Ransom	Intestine
Centrocestus cuspidatus Looss (*var.* caninus Leiper)	Intestine
Cryptocotyle lingua (Creplin) [=Hallum caninum Wigdor]	Intestine
Paragonimus westermanii (Kerbert)	Lungs
Paragonimus kellicotti Ward	Lungs
Schistosoma japonicum Katsurada	Veins
Alaria alata (Dies.)	Intestine
Alaria americana Hall and Wigdor	Intestine
Alaria michiganensis Hall and Wigdor	Intestine

3. *Sus scrofa* Linn. Pig

Echinochasmus perfoliatus Ràtz	Intestine
Fasciola hepatica (Abildg.)	Liver
Fasciolopsis buskii (Lankester)	Intestine
Dicrocoelium lanceatum St. and H.	Gall-bladder
Loxotrema ovatum Kob.	Intestine
Paragonimus westermanii Kerbert	Lungs
Microtrema truncatum Kobayashi	Liver
Distomum suis Stiles (larva)	Muscles in cysts
Gastrodiscus minor Leiper	Liver and intestine
Gastrodiscoides hominis Lewis and McConnell	Intestine

4. *Bos taurus* Linn. Ox

Fasciola hepatica (Abildg.)	Liver and gall-bladder
Fasciola gigantica (Cobbold)	Liver
Fascioloides magna (Bassi)	Liver and lungs
Dicrocoelium lanceatum St. and H	Liver and gall-bladder
Dicrocoelium hospes Looss	Bile ducts
Eurytrema coelomaticum Giard and Billet	Pancreatic duct

4. *Bos taurus* Linn. (*continued*)

Eurytrema pancreaticum Janson	Pancreatic duct
? Eurytrema parvum Senoo	Pancreas
Schistosoma bovis Sonsino	Ureters, bladder and veins
Schistosoma spindalis Montgomery	Veins
Paramphistomum cervi (Schrank)	Stomach
Paramphistomum calicophorum Fischoeder	Stomach
Paramphistomum explanatum (Creplin)	Gall-bladder, bile duct
Paramphistomum scoliocoelum Fischoeder	Stomach
Paramphistomum tuberculatum (Cobbold)	Stomach
Cotylophoron cotylophorum Fischoeder	Stomach
Verodunia tricoronata Lahille and Joan	Stomach
Stephanopharynx compactus Fischoeder	Stomach
Gastrothylax crumenifer (Creplin)	Stomach
Gastrothylax compressus Brandes	Stomach
Fischoederius cobboldi (Poirier)	Stomach
Fischoederius elongatus (Poirier)	Stomach
Carmyerius spatiosus (Brandes)	Stomach
Carmyerius mancupatus (Fischoeder)	Stomach
Carmyerius gregarius (Looss)	Stomach
Homalogaster poirieri Giard and Billet	Colon

5. *Ovis aries* Linn. Sheep

Fasciola hepatica (Abildg.)	Liver
Fasciola gigantica (Cobbold)	Liver
Fascioloides magna (Bassi)	Liver and lungs
Dicrocoelium lanceatum St. and H.	Liver and gall-bladder
Schistosoma bovis Sonsino	Portal veins
Paramphistomum cervi (Schrank)	Stomach
Cotylophoron indicum St. and H.	Stomach

6. *Capra hircus* Linn. Goat

Fasciola hepatica (Abildg.)	Liver
Paramphistomum cervi (Schrank)	Stomach

7. *Equus asinus* Linn. Ass

Fasciola hepatica (Abildg.)	Liver
Schistosoma indicum Montgomery	Veins

8. *Equus mulus* Linn. Mule

Gastrodiscus secundus Looss	Intestine

9. *Equus caballus* Linn. Horse

Fasciola hepatica (Abildg.)	Liver
Fascioloides magna (Bassi)	Liver
Pseudodiscus collinsii (Cobbold)	Large intestine
Pseudodiscus stanleyii (Cobbold)	Large intestine
Gastrodiscus aegyptiacus (Cobbold)	Coecum and colon

LIST OF BRITISH MAMMALS FROM WHICH NO TREMATODE PARASITES HAVE HITHERTO BEEN RECORDED.

1. *Muscardinus avellinarius* Linn.	Dormouse
2. *Micromys minutus* Pall.	Harvest mouse
3. *Apodemus hirtensis* Linn.	St Kilda long-tailed field mouse
4. *Apodemus flavicollis* Melch.	Yellow-necked mouse
5. *Mus muralis* Barrett and Hamilton	St Kilda Island mouse
6. *Mus rattus* Linn.	Black rat
7. *Microtus orcadensis* Millais	Orkney Island vole

8.	*Microtus agrestis* Linn.	Field vole
9.	*Microtus sandayensis* Millais	Sandy Island vole
10.	*Evotomys glareolus* Schreb.	Bank vole
11.	*Evotomys skomerensis* Barrett and Hamilton	Skomer Island Bank vole
12.	*Lepus europaeus* Pall.	Common hare
13.	*Lepus hibernicus* Bell	Irish hare
14.	*Mustela erminea* Linn.	Stoat
15.	*Mustela hibernica* Thomas and Hamilton	Irish stoat
16.	*Phoca hispida* Schreb.	Ringed seal
17.	*Cystophora cristata* Erxl.	Hooded seal
18.	*Physeter macrocephalus* Linn.	Sperm whale
19.	*Mesoplodon bidens* Sowerby	Sowerby's whale
20.	*Hyperoodon rostratus* Müll.	Bottle-nosed whale
21.	*Monodon monoceros* Linn.	Narwhal
22.	*Delphinapterus leucas* Pall.	Beluga
23.	*Orcinus orca* Linn.	Killer
24.	*Globicephalus melas* Traill	Pilot whale
25.	*Grampus griseus* Cuv.	Risso's grampus
26.	*Lagenorhynchus albirostris* Gray	White-beaked dolphin
27.	*Lagenorhynchus acutus* Gray	White-sided dolphin
28.	*Balaena glacialis* Bonn.	Black right whale
29.	*Megaptera nodosa* Bonn.	Hump-backed whale
30.	*Balaenoptera musculus* Linn.	Sibbald's rorqual
31.	*Balaenoptera physalus* Linn.	Common rorqual
32.	*Balaenoptera acutorostrata* Lac.	Lesser rorqual
33.	*Sorex minutus* Linn.	Pigmy shrew
34.	*Barbastella barbastellus* Schreb.	Barbastelle
35.	*Myotis bechsteini* Kuhl	Bechstein's bat

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A PROPOS DES IXODIDÉS DE RUSSIE.

PAR LE PROFESSEUR W. L. YAKIMOFF.

Du Service de Protozoologie de l'Institut Vétérinaire Bactériologique de Pétrograde.

Nous avons décrit dans le *Bulletin de la Société de Pathologie Exotique* (t. XV, séance du 11 Janvier 1922) les genres et les espèces d'Ixodidés de Russie, la distribution géographique des tiques, leurs hôtes et parmi eux les vecteurs de parasites sanguicoles. 37 espèces réparties en 8 genres: 9 du genre *Ixodes* (dont 5 douteuses), 4 du genre *Rhipicephalus*, 1 du genre *Boophilus*, 4 du genre *Hyalomma*, 1 du genre *Aponomma* (douteuse), 3 du genre *Dermacentor*, 8 du genre *Haemaphysalis*, 3 du genre *Argas* et 3 du genre *Ornithodorus*.

Nous croyons qu'il est utile de donner les clefs pour les genres et pour les espèces des tiques de Russie:

I. LES GENRES.

1. Pas d'yeux 2
   Des yeux 3
2. Rostre long . . . . . *Ixodes*
   Rostre court . . . . . *Haemaphysalis*
3. Rostre long . . . . . *Hyalomma*
   Rostre court 4
4. Face supérieure de la base du rostre héxagonale. Mâle avec écussons adanaux . 5
   Face supérieure de la base du rostre quadrangulaire. Mâle sans écussons adanaux *Dermacentor*
5. Stigmates en virgules. Sillon contournant l'anus postérieurement . *Rhipicephalus*
   Stigmates ovales ou circulaires. Pas de sillon anal . . . . *Boophilus*

II. LES ESPÈCES.

Genre IXODES.

Mâle.

Hanche I à une épine interne forte, atteignant hanche II. Tarses sans protubérance dorsale. Sillon anal à branches divergentes . . . . . *ricinus*

Hanche I à une épine interne courte. Tarses avec protubérance dorsale subterminale. Sillon anal à branches parallèles . . . . . *hexagonus*[1]

[1] Quoique l'*Ixodes hexagonus* jusqu'à présent ne se rencontre pas en Russie nous le plaçons ici parce que la zone géographique de la répartition de l'*Ixodes ricinus* au nord de Russie est voisine de Norvège.

Femelle.

1. Hanche I inerme 2
   Hanche I épineuse 3
2. Palpes longues, cultriformes; 1er article en forme de corne transversale. Tarses sans protubérance dorsale . . . . . *trianguliceps*
   Palpes courtes. 1er article sans prolongement. Tarses pourvus d'une protubérance dorsale subterminale . . . . . *hirsutus*
3. Hanche I à une épine interne 4
   Hanche I à deux épines courtes . . . . . *berlesei*
4. Hanche I à une épine interne courte. . . . . *hexagonus*
   Hanche I à une épine longue 5
5. Ecusson dorsal pourvu d'un sillon latéral . . . . . *ricinus*
   Ecusson dorsal dépourvu d'un sillon latéral . . . . . *spinicoxalis*

Genre Rhipicephalus.

Mâle.

1. Ecusson dorsal à ponctuations très inégales 2
   Ecusson dorsal à ponctuations égales . . . . . *simus*
2. Sillons marginaux limitant les 2 festons extrêmes ou se terminant entre eux . . 3
   Sillons marginaux limitant un feston extrême . . . . . *bursa*
3. Sillons marginaux limitant les 2 festons extrêmes. Angle extérieur de la base du rostre pas aigu et quelquefois émoussé. Ecussons adanaux triangulaires; angle postéro-extérieur obtus. Péritrèmes étroits . . . . . *sanguineus*
   Sillons marginaux entre 2 festons extrêmes. Angle postérieur de la base du rostre très aigu. Ecussons adanaux quadrangulaires; angle postéro-extérieur plus droit. Péritrèmes plus larges . . . . . *rossicus*

Femelle.

1. Aires poreuses ovales ou rondes 2
   Aires poreuses presque rondes ou angulaires 3
2. Sillons latéraux indiqués chacun par une ligne de grosses ponctuations . *simus*
3. Ecusson dorsal oval allongé, un peu plus long que large. Aires poreuses peu écartées; péritrèmes étroits; angle postérieur aigu . . . . . *sanguineus*
   Ecusson dorsal plus arrondi; longueur = largeur. Aires poreuses très écartées. Péritrèmes plus larges; angle postérieur arrondi . . . . . *rossicus*

Genre Dermacentor.

1. 2ième article des palpes plus large que long avec une épine rétrograde . . . 2
   2ième article des palpes à peine ou pas plus large que long, sans épine . . *variegatus*
2. 2ième article des palpes avec une épine rétrograde courte . . . . *niveus*
   2ième article des palpes avec une épine rétrograde plus longue. Taille un peu plus petite que dans l'espèce précédente . . . . . *reticulatus*

Genre Hyalomma.

1. Hanche I divisée peu profondément . . . . . *syriacum*
   Hanche I divisée profondément 2
2. Ecusson dorsal pourvu de ponctuations profondes et nombreuses. Sillon latéral . 3
   Ecusson dorsal pourvu de ponctuations rares, superficielles. Pas de sillon latéral *rhipicephaloides*
3. Ecussons accessoires rectangulaires . . . . . *aegyptium*
   Ecussons accessoires recourbés en pointe . . . . . *dromedarii*

Genre HAEMAPHYSALIS.

Mâle.

1. Palpes à 3ième article formant pince avec son congénère . . . . . *concinna*
   Palpes à 3ième article ne formant pas de pince 2
2. Hanche IV à une épine aussi longue ou presque aussi longue que large . *punctata*
   Hanche IV à une épine courte ou nulle 3
3. Palpes à 3ième article pourvu d'une courte épine . . . . . *papuana*
4. Hypostome à 10 (rarement 12) files de dents. Sillon latéral long, terminé entre les festons extrêmes et pénultièmes . . . . . *neumanni*
   Hypostomes à 6 files de dents. Sillon latéral court, arrêté à la limite antérieure du feston extrême . . . . . *leporis*

Femelle.

1. Palpes à 3ième article pourvu d'une épine ventrale rétrograde 2
   Palpes à 3ième article sans épine ventrale rétrograde 5
2. Palpes à 3ième article sans épine dorsale rétrograde 3
   Palpes à 3ième article pourvu d'une épine dorsale rétrograde 4
3. Palpes à 2ième article arrondi en dehors. Hanche I et IV à épines égales . *punctata*
   Palpes à 2ième article anguleux. Hanche I à épine longue. Hanche IV à épine très courte . . . . . *concinna*
4. Ecusson dorsal un peu retréci en arrière et un peu anguleux . . *neumanni*
   Ecusson dorsal ovale . . . . . *leporis*
5. 2ième article des palpes anguleux en dehors. Hypostome à 10 files de dents *papuana*
   2ième article des palpes non saillant en dehors. Hypostome à 6 files de dents *ambigua*

Genre ARGAS.

1. Corps discoïde (aussi large devant que derrière) . . . . . *vespertilionis*
   Corps ovale (plus long que large, plus étroit en avant) 2
2. Bordure du corps formé de festons rectangulaires . . . . . *persicus*
   Bordure du corps formé de plis étroits . . . . . *reflexus*

Genre ORNITHODORUS.

1. Camerostome pourvu d'ailes latérales . . . . . *talaje*
   Camerostome dépourvu d'ailes latérales 2
2. Tarses I à 3 tubercules dorsales . . . . . *canestrinii*
   Tarses I à saillie dorsale subterminale . . . . . *tholozani*

A PROPOS DU *RHIPICEPHALUS SANGUINEUS* ET *RHIPICEPHALUS ROSSICUS.*

PAR LE PROFESSEUR W. L. YAKIMOFF.

Du laboratoire protozoologique de l'Institut vétérinaire-zootechnique de Pétrograde.

Nous avons étudié avec Mme Nina Kohl-Yakimoff en 1911[1] au laboratoire de Mr le Professeur Neumann (à Toulouse) une tique russe recueillie sur des chiens dans le gouvernement de Saratov. Nous l'avons estimée comme une espèce nouvelle et l'avons nommée *Rhipicephalus rossicus*. Dans notre note nous avons décrit les différences entre cette tique et l'espèce voisine *Rhipicephalus sanguineus* Latr., 1806.

Actuellement nous donnons quelques nouveaux détails qui confirment cette différence.

Rhipicephalus sanguineus	*Rhipicephalus rossicus*
Mâle.	
Ecusson dorsal	
Ne couvre pas une petite bordure sur les côtés et en arrière du dos.	Couvre toute la surface du dos et seulement quelquefois ne couvre entièrement.
Angle extérieur de la base du rostre	
Pas aussi aigu et quelquefois émoussé.	Très aigu et très saillant.
Sillons et fossettes	
A la partie postérieure du corps un sillon médian avec une fossette de chaque côté. Sillons marginaux limités par deux festons extrêmes.	Au lieu du sillon médian une fossette ovale allongée de chaque côté de laquelle se trouve une fossette ronde. En avant presque au milieu du corps encore deux autres fossettes rondes. Sillons marginaux entre les fossettes extrêmes.
Sillons adanaux	
Triangulaires. Le bord intérieur droit. Angle postérieur externe obtus.	Quadrangulaires. Le bord intérieur divisé en deux moitiés inégales: l'intérieure plus grande et la postérieure plus petite, droite ou presque droite. Angle postérieur externe plus droit.
Hanches	
Hanche I divisée en 2 épines longues. Hanches II–IV avec 2 épines courtes.	Hanche I & IV divisées en 2 épines longues. Hanche II & III avec deux épines courtes.
Péritrèmes	
Etroits. Angle postérieur aigu.	Plus larges. Angle postérieur arrondi.

[1] W. L. Yakimoff et Nina Kohl-Yakimoff (1911). Étude des Ixodidés de Russie. *Archives de Parasitologie*, XIV, p. 146.

Femelle.

Ecusson dorsal	
Oval allongé. Longueur > largeur.	Plus arrondi. Longueur = largeur.
Sillons cervicaux	
Continus.	Leur trajet interrompu deux fois.
Sillons latéraux	
Longs. Se rencontrent presque avec des sillons cervicaux près du bord postérieur.	Courts. Ne parviennent pas au bord postérieur et ne se rencontrent pas avec sillons cervicaux.
Palpes	
II article un peu anguleux.	II article arrondi.
Yeux	
Au milieu de la longueur de l'écusson ou un peu en arrière.	Toujours au milieu.
Aires poreuses	
Larges. Elliptiques. Peu écartées.	Forme irrégulière. Triangulaires. Très écartées.
Péritrèmes	
Etroits. Angle postérieur aigu.	Plus larges. Angle postérieur arrondi.

FURTHER INQUIRIES INTO THE ZOOLOGICAL STATUS OF THE POLYMORPHIC MAMMALIAN TRYPANOSOMES OF AFRICA, AND THE MEANS BY WHICH THEY ARE SPREAD IN NATURE.

By H. LYNDHURST DUKE, O.B.E., M.D., Sc.D. (Cantab.).

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INTRODUCTION.

The object of this paper is to develop the ideas put forward in previous publications (Duke, 1919, 1921).

The paper is divided into two parts. Part I sets forth the continuation of experiments, commenced in 1920, on the effect of continued direct transmission on a strain of polymorphic trypanosomes originally recovered from wild lake-shore *Glossina palpalis* on Victoria Nyanza. The earlier stages of these experiments have already been published (Duke, 1921). Similar experiments with a strain of polymorphic trypanosomes recovered in September 1920 from the blood of an antelope shot on Damba Island, Victoria Nyanza, are given for comparison.

In Part II an attempt is made to discuss the Trypanosomiasis Problem in the light of the theories put forward in the two papers just referred to. It is

hoped that the importance and urgency of the issue will be held to counterbalance the presumption shown in attempting so ambitious a task. If the contentions here advanced are correct, it will be necessary to revise our views regarding the nature and spread of human trypanosomiasis, and on the relation of the Big Game of Africa to Man. It is believed, however, that the situation will thereby assume a more hopeful aspect, and the problem of control will be greatly simplified.

It is not expected that the interpretations here put forward will meet with immediate or general acceptance. They challenge several firmly established notions. But they contain enough solid foundation to warrant critical investigation; and this paper will have served its purpose if it stimulates free discussion of the doctrines it supports.

Throughout the paper, the word "game" implies the Big Game, as generally understood, and not small animals and birds. By "game-tsetses" are understood, more especially, *G. morsitans* and *G. pallidipes*. The expressions "proboscis-only," "proboscis-and-gut," and "gut-and-gland" groups, denote the three natural divisions into which the tsetse-carried trypanosomes fall, according to their developmental behaviour in the *Glossina* intermediary.

In the course of this paper frequent reference is made to an outbreak of human trypanosomiasis in a "*morsitans* belt" in the neighbourhood of Mwanza, in Tanganyika Territory. The report of a six-weeks' tour in this infected area has been published elsewhere (Duke, 1923). For convenience, a brief recapitulation will be given here.

The disease was first recognized by the natives of the fly-belt about two years ago, and is considered by them to be new. The tsetse concerned with the spread of the trypanosome is *G. swynnertoni* Austen, 1923. The causative trypanosome is indistinguishable from the *Trypanosoma brucei* found in both game and fly in the belt: it produces posterior-nuclear forms in rats and guinea-pigs, causes keratitis in dogs, and has its anterior station in the salivary glands of the tsetse.

The two human strains from this belt that have hitherto been investigated develop readily in laboratory-bred *G. palpalis*, flagellates being present in the gut and in the salivary glands. The average period elapsing before the fly becomes infective is 21·6 days (minimum 18 days, maximum 26 days).

PART I. EXPERIMENTAL.

A STUDY OF THE EFFECTS PRODUCED ON A TRYPANOSOME STRAIN BY MAINTENANCE FOR TWO YEARS AND NINE MONTHS BY DIRECT TRANSMISSION.

(1) The strain of trypanosomes selected for these experiments was one of the gut-and-gland group of organisms, derived from wild *G. palpalis* from the mainland shore of Victoria Nyanza, near Entebbe. The strain was first recovered in January 1920, in monkey 43, upon which the flies were originally fed.

The duration of the disease in this monkey was 63 days.

From monkey 43, a second monkey, No. 59, was infected by cyclical passage of the strain through laboratory-bred flies.

Starting from monkey 59, a series of passages by direct transmission was carried out.

Table I shows these experiments in detail.

Table I. *Showing the maintenance of the Direct-Transmission Strain from January* 1920 *until October* 1922.

Experiment No.	No. of passage	Duration of disease in days	Day of disease in infecting animal when sub-inoculation performed
+ 59		407	flies fed on monkey 43 from 9th to 17th days of its infection
+ *72*	1	77	about *20*
+ *88*	2	Alive after 449 days	„ *29*
+ *98*	3	Still alive after 917 days	„ *20*
+ *107*	4	*140*	„ *9*
+ *111*	5	*90*	„ *9*
+ *117*	6	*125*	„ *10*
+ *123*	7	?	„ *17*
+ *132*	8	*254*	„ *18*
+ *140*	9	*94*	„ *9*
+ *147*	10	*92*	„ *26*
161	11	*246*	*55*
172	12	*91*	*119*
293	13	*140*	*60*
295, *300*	14	62, *47*	31, *102*
304	15	*10*	*38*
305	16	*37*	*9*
306, *311*, 310	17	38, *37*, 11	6, *24*, 14
313	18	*28*	*11*
316	19	*8*	*21*
317	20	*7*	*8*
319	21	*7*	*7*
320, *321*	22	10, *24*	7, *7*
325, *324*	23	10, *38*	12, *12*
326	24	*18*	*13*
329	25	*8*	*17*
330, *331*	26	9, *9*	7, *7*
332	27	*12*	*9*
333	28	*23*	*12*
336	29	*19*	*12*
337	30	*19*	*14*
338	31	*16*	*7*
341	32	*15*	*13*
355	33	*15*	*12*
345	34	*11*	*21*
396, *397*	35	19, *30*	6, *6*
G. pigs 419, *420*, 430	36	21, *50*, 27	11, *11*, 18
445	37	*21*	*31*
447	38	*24*	*13*
G. pig 477, monkey *478*	39	18, *10*	12, *12*
500	40	*27*	*10*
509	41	*26*	*5*
G. pig 525	42	*19*	6

In experiments marked + infection was effected by means of interrupted fly-feedings.

To effect the earlier passages, clean laboratory-bred *G. palpalis* were used, infection being secured by interrupted feeding of the flies first on the infected and then immediately on the clean animal.

This method was tedious, and occupied a great deal of time, and accordingly the fly-method was given up at the 11th passage for the inoculation of a small quantity of citrated blood from the sick to the healthy monkey.

As the strain became more virulent it was necessary occasionally to introduce a guinea-pig into the series, as monkeys were not always available.

At the 42nd passage, a series of unavoidable accidents led to the loss of the strain (October 1922).

The same species of monkey, a greenish grey *Cercopithecus* with a black face, was employed throughout.

The monkeys taken for these experiments were healthy and well-fed. Nos. 317 and 319 were big powerful animals, and I have often noticed that monkeys of this type show far less resistance in captivity than smaller and gentler animals.

In Table I the number of the animal from which the serial sub-inoculation was made is in italics, as are also the corresponding figures in the other columns. Numbers not in italics indicate duplicate inoculations which were not continued.

Apparently the virulence of this strain began to increase at the 15th passage.

It will be seen that in the majority of the passages shown in Table I the trypanosomes were transferred from the infected to the healthy monkey early in the disease. In this respect these experiments differ somewhat from those set forth in Table IV.

Experiments with laboratory-bred G. palpalis *and the later passages of the "Direct-Transmission" Strain.*

Technique. G. palpalis, bred in the laboratory, were placed in wire-sided boxes and fed on the infected monkey under experiment.

In the experiments of Table I, which were started before I had any suspicion that a change had occurred in the transmissibility of the strain, the flies, after the infecting feedings, were nourished on cock's blood for 2 or 3 weeks and then tested on a clean monkey. This was done to economize monkeys.

Tables II and III show these transmission experiments in detail. The experiments of Table I were carried to full term, as it was intended to see whether cyclical transmission of this virulent strain led to any reduction in its virulence.

When it became evident that the strain had lost the power of developing in the fly, the duration of the experiments was shortened to about 14 days; for within this period any developing infections would be easily recognizable, and longer manipulation of negative boxes was a waste of time.

The day of the disease in the monkey is given because it indicates the stage in the infection at which the flies were put on.

In case the cock's blood might have exerted an inhibitive effect on the flagellates in the first series, the flies in the experiments of Table III were nourished throughout on monkey's blood.

Table II.

Expt. No.	Infecting monkey	Dissection began on day	No. of flies alive on 25th day	Flies dissected			Duration of expt. in days	Day of disease in infecting monkey when flies put on
				Males	Females	Total		
387	345	18	49	31	21	52	52	11
388	381	15	39	29	15	44	50	5
394	381	8	41	36	14	50	45	8
401	345	11	40	29	21	50	48	16
407	396	11	75	28	51	79	55	3
408	396	10	54	23	33	56	51	3
409	397	11	48	22	28	50	55	3
410	345	9	51	27	36	63	55	20
412	397	8	46	34	29	63	58	5
414	396	9	42	25	30	55	56	6
415	396	10	49	33	37	70	52	7
416	397	9	54	25	42	67	53	8
417	396	8	42	22	27	49	57	9
421	397	10	56	25	40	65	51	11
423	396	10	65	46	36	82	54	12
428	396	9	46	20	33	53	45	17
		Totals	797	455	493	948		

Table III.

Expt. No.	Infecting monkey	Dissection began on day	No. of flies alive on 13th day	Flies dissected			Duration of expt. in days	Day of disease in infecting monkey when flies put on
				Males	Females	Total		
495	447	14	45	28	18	46	19	20
501	447	18	52	24	28	52	18	22
805	500	6	49	42	14	56	14	4
508	500	7	62	32	33	65	14	5
512	500	13	53	30	23	53	13	7
514	500	15	60	24	36	60	15	8
816	500	10	68	45	23	68	14	9
517	509	14	51	24	27	51	14	4
523	500	14	65	21	44	65	14	11
537	509	8	51	22	32	54	17	12
		Totals	556	292	278	570		

In not a single one of the flies used in these experiments were any flagellates found.

That these consistently negative results were not due to any peculiar climatic conditions at the laboratory is shown by the fact that, in transmission experiments carried out at the same time with other gut-and-gland strains, a large number of positive flies was obtained.

It is evident from these experiments that this strain has completely lost the power of developing cyclically in the Glossina *from which it was originally recovered.* At what stage in the series of passages this power was lost, it is impossible to say. The last positive evidence was obtained at the 11th passage when a fly became infective 31 days after feeding on the monkey. By the 34th passage the power to survive in the fly had been lost.

(2) We will now consider the behaviour of the "antelope-strain" under similar experimental manipulations.

This strain was obtained from an antelope shot on Damba Island, Victoria Nyanza, and is still being maintained at the laboratory, but has not yet shown any striking enhancement of virulence. Table IV gives the history of its upkeep.

Table IV. *Showing the maintenance, by sub-inoculation with the syringe, of the Antelope Strain, from September* 1920 *until November* 1922.

Experiment No.	No. of passage	Duration of disease in days	Day of disease when monkey sub-inoculated
173 (Inoculated from wild antelope)	1	66	—
171	2	38	*34*
G. pig *198*	3	47	*40*
Dog *232*	4	25	*47*
275 (Infected by cyclically infected lab.-bred flies off dog 232)	5	78	(13–15th)
290	6	28	*76*
291	7	28	*18*
292	8	33	*10*
294, 296	9	*43*, 36	*21*, 33
298, 297	10	*50*, 17	*41*, 34
301, 299	11	*20*, 23	*37*, 14
302	12	37	*14*
303	13	34	*9*
308, 307	14	*36*, 19	*31*, 27
312	15	23	*14*
314	16	45	*20*
318, 315	17	*23*, 4	*24*, 20
323, 322	18	*34*, 34	*11*, 11
327, 328	19	*57*, 38	*33*, 33
334	20	*34*	*35*
335	21	*42*	*12*
339, 350	22	*29*, 11	*23*, 41
366, 365	23	*22*, 20	*29*, 29
389	24	*24*	*20*
G. pigs *418*, 431	25	*56*, 53	*12*, 22
G. pig *437*, monkey 444	26	77, 16	*25*, 32
G. pigs *510*, 565, monkey 446	27	*44*, 28	*47*, 13
G. pig 566, monkeys 567, *568*, 448	28	16, 16, *30*, 20	39, 41, *41*, 7
581	29	—	15

Tables I and IV supply no obvious explanation of the difference in the behaviour of the two strains. In Table I the early passages were effected more rapidly than was the case in the experiments recorded in Table IV. On the other hand, the alteration in virulence was first manifested after a consecutive series of six slow passages, monkey 147 being infected about 4. viii. 20 and monkey 304 on 24. x. 21—a period of 446 days.

All the passages were made by means of the syringe, save the 5th which was effected by laboratory-bred flies cyclically infected from dog 232.

Reckoning, therefore, from the infection of monkey 290 from monkey 275, this antelope-strain has undergone 24 direct passages between 9. v. 1921 and 9. xi. 1922.

Turning now to the behaviour of this strain in laboratory-bred *G. palpalis* as set forth in Table V, we see that interesting changes have occurred in the strain since it was first recovered from the blood of the antelope in September 1920.

Table V.

Experiment No.	Infecting monkey	Day on which dissection of flies began	No. of flies alive on 25th day	Flies dissected			Duration of experiment (days)	Day of disease in infecting monkey when flies put on	Positive flies
				Males	Females	Total			
570	567	8	31	10	36	46	37	8–10	0
571	568	5	47	35	21	56	41	9–11	1
572	567	8	36	22	21	43	41	10–12	2
574	568	13	28	26	21	47	39	12–14	0
575	567	15	46	24	25	49	56	13–15	0
579	568	11	44	20	31	51	42	14–16	1
580	567	9	44	30	34	64	54	15–17	2
589	581	12	51	20	42	62	50	5– 7	3
590	568	10	52	39	17	56	48	22–24	2
591	581	16	47	28	22	50	48	7– 9	1
		Totals	426	254	270	524			12

Table VI. *Positive flies from the experiments shown in Table V.*

Expt. No.	No. of fly containing flagellates	Intensity of infection		Day of experiment on which fly was dissected
		Gut	Glands	
571	1	+ + +	–	10
572	2	+ + +	–	12
,,	3	+	–	41
579	4	+ +	–	42
580	5	+ + +	–	50
,,	6	+	–	51
589	7	+	–	38
,,	8	+	–	46
,,	9	+ +	–	50
590	10	+ + +	–	21
,,	11	+ +	–	48
591	12	+ + +	–	48

From Table V it will be seen that flagellates were found in 2·2 per cent. of the flies employed. Table VI, which shows the results of the dissections of the positive flies in these experiments, reveals the interesting fact that *in no case were flagellates found in the salivary glands*, and thus explains why all these transmission experiments were negative.

Previous transmission experiments with this antelope-strain and laboratory-bred *G. palpalis*, carried out between 5. x. 20 and 26. i. 21, showed that flagellates developed in 2·32 per cent. of the flies, but that the percentage of *infective* flies was 0·68—this latter figure being higher than that obtained with either the human or the wild-fly strains with which similar experiments were carried out more or less concurrently (Duke, 1921). The average duration of the full developmental cycle with the newly isolated antelope-strain was 28 days (maximum 40, minimum 19 days).

It is clear, therefore, that an important change has occurred in the strain between January 1921 and November 1922 when the experiments set forth in Table V were commenced. *The percentage of positive flies is about the same, but the percentage of infective flies is now nil.*

It is possible that, if the experiments had been prolonged, infection of the salivary glands might have occurred; on the other hand, Table VI shows that in nine of the flies the average, and in the eight the maximum period

that in 1920 was necessary for the completion of the full cyclical development, was exceeded, and yet no invasion of the glands had occurred. Again, the intensity of the gut-infection shows a curious irregularity, flies nos. 1 and 2 of Table VI having heavy gut-infections, while nos. 3, 6, 7 and 8, all old flies, showed very small infections. Apparently the power of the trypanosome to undergo full cyclical development in the fly has been seriously impaired if not completely lost.

The fact that this antelope-strain can still produce massive gut-infections, but is unable to infect the salivary glands, is suggestive. Apparently this state of affairs is intermediate between the normal behaviour of the strain when newly isolated from its natural environment, and the end-product of prolonged direct transmission as exemplified by the Direct-Transmission strain of Table I.

It is difficult to decide whether the complete loss of the power to render the fly infective is intimately associated with the acquisition by the strain of increased virulence. Table VI suggests that the process of invasion of the salivary glands by the trypanosome is considerably more intricate and complex than the establishment and multiplication of the flagellates in the gut.

How the invasion of the glands is effected is not known for certain. Miss Robertson believes that the invasion of the salivary glands by the trypanosome is a mere forward migration of flagellates situated in the anterior gut. Whether this migration is dependent on a certain degree of infestation of the gut with flagellates, or is brought about by the development, by sexual processes, of certain special invading types, we do not know.

The failure of this strain to negotiate the final and essential stages of the cycle in the fly, seems to me to point to the development being asexual rather than sexual in nature. One would imagine, if a sexual development could proceed so far as to produce a + + + infestation of the gut, that invasion of the glands would follow in due course; whereas if the whole process is asexual, and invasion of the glands only occurs after a certain intensity of gut infestation is reached, then it is comprehensible that impairment of the cultivability of the trypanosome would render its development abortive.

If a conclusion is justifiable from the limited evidence set forth in Part I it is that continued upkeep by direct transmission leads first of all to the loss by the trypanosome of the power of invading the salivary glands, and this is followed later, in the history of the strain, by the complete loss of the power to develop in any part of the alimentary canal of the insect. Further, I think that the evidence we have before us tends to support the view that the cyclical development of the trypanosome in the fly is not a sexual process.

Miss Robertson records an instance of a fly showing a massive gut-infection 56 days after its infecting feed, but no flagellates in the salivary glands; this, however, was noted as a peculiar phenomenon occurring in the course of a long series of experiments with readily transmissible strains. This observation shows, however, that the invasion of the salivary glands may be postponed

greatly beyond the normal period, even in cyclically transmitted strains, and it is quite possible that in the case quoted above the fly might never have become infective.

It should be added that both the "direct-transmission" and the "antelope" strains retained their polymorphism throughout the experiments reported above.

Applying these results to the spread of trypanosomes in human communities, we should expect direct transmission to function for a time without producing any noticeable effect on the strain so transmitted. Sooner or later, if conditions were favourable, the virulence of the parasite would increase, and direct transmissions would, in consequence, become increasingly frequent.

As the virulence increased the trypanosome would lose its power of cyclical development in the tsetse, until it came to rely, for its transmission from man to man, entirely on the direct method.

In such circumstances the epidemic would continue for just so long as the conditions admitted of spread by the direct method. When this became no longer possible the strain would presumably die out.

Probably, in actual life, the enhancement of the virulence of a strain of human trypanosomes only occurs where the contact between fly and man is very intimate, and the passage of the parasite from host to host, in consequence, very rapid[1].

PART II. ANALYTIC.

The polymorphic mammalian trypanosomes whose development in *Glossina* has been worked out, are, by general consent, divided into two categories, each comprising several so-called species. On the one hand are *T. gambiense* and *T. nigeriense*, originally described from man, *T. multiforme* of Kinghorn and Yorke (Kinghorn, 1913), Balfour's mule trypanosome from the Sudan (Balfour, 1913), and the organisms isolated from domestic animals in Principe. The animal reactions of these strains are chronic, and the development of posterior-nuclear forms has not been recorded. In the other category are *T. brucei*, *T. rhodesiense*, and a number of similar organisms isolated by different people from game and stock in various parts of Africa. These strains are characterized by their greater virulence and by the formation of posterior-nuclear forms.

Investigators are specially concerned with the trypanosomes that affect man; and the mere fact that it can survive in man has been regarded as sufficient justification for the creation of a new species in *T. rhodesiense*.

In studying all these trypanosomes one cannot fail to be struck by the strong resemblances among the so-called species. Morphologically, they

[1] I have much pleasure in acknowledging my indebtedness to Miss Mary Martin, M.B., D.P.H., Assistant Bacteriologist, Uganda, for invaluable help in the conduct of these experiments. My thanks are also due to Mr E. C. Haddon, Laboratory Assistant, in whose hands rested the upkeep of the Direct Transmission and the Antelope Strains during my absence on leave, and to Mr J. Stewart, Laboratory Assistant, who has looked after the strains since his return from leave.

exhibit no fixed differences that a zoologist would consider of specific value: the development in the fly, of those strains that have been thoroughly investigated, is the same, the "anterior station" being in the salivary glands.

The response to human serum, and cross-immunity reactions, upon which considerable stress has been laid in subdividing the group, are tests which have of late fallen into discredit with those who have had most experience in their employment.

The two categories are generally distinguished from one another by differences in virulence, and by the presence or absence of what are known as "posterior-nuclear" forms.

(1) *Posterior-nuclear forms.*

These forms are said not to be found in *T. gambiense*, while they always occur in rats inoculated with members of the *brucei* group. They have been seen by Wenyon (1912) in "*T. pecaudi*" in the Sudan, by Yorke and Blacklock (1912) in *T. equiperdum*, by Blacklock (1912) in *T. brucei*, and by many other observers. There is little doubt that the occurrence of this peculiarity is intimately connected with virulence.

It is generally stated that posterior-nuclear forms are seen only in certain laboratory animals, in which the trypanosome causes a rapidly fatal disease, and that they are most in evidence when the parasites are swarming in the blood.

Mr Richardson, the Veterinary Pathologist, Uganda, recently called my attention to a most interesting case of cattle trypanosomiasis from the Sese Islands, in which we found posterior-nuclear forms in the peripheral blood. The animal—an ox—was infected with a member of the polymorphic group, and also with *T. vivax*, and (probably) *T. uniforme*. It had been infected by wild *G. palpalis*. The slide examined showed a very large number of parasites in the peripheral blood, and among them were typical posterior-nuclear forms with the two nuclei lying close together, and also a number of almost equally pronounced anterior-nuclear forms. There is no doubt that the multiple infection with two (or possibly three) trypanosomes had completely upset the animal's resistance. As a result, the trypanosome, unrestrained by the normal resistance of its host, manifested the phenomena usually associated with its presence in rats—namely, rapid multiplication in the peripheral blood, together with the morphological distortions that accompany such rapid proliferation.

The animals in which this phenomenon is best seen—rats and guinea-pigs—cannot possibly be regarded as natural hosts of the trypanosome; they are constitutionally unsuited to its propagation, and die in a very short time of a fulminant disease.

It is interesting to note Wade Brown's remarks on the relation of morphological abnormalities to virulence in *T. lewisi*. He says: "The control strains, which, when first isolated, produced very benign infections and showed no

anomalies, subsequently showed an increased virulence in certain series of infections coincident with the appearance of anomalous forms of trypanosomes in the blood of infected rats." Again, paragraph 3 of the summary reads: "It is pointed out that the morphological anomalies were most pronounced in infections that showed unusual conditions of multiplication, and that such infections usually proved severe" (Wade Brown, 1914).

It is evident from what has been said above that it is unjustifiable and misleading to endow this character with specific value. The separation of *T. brucei* from *T. gambiense* on such grounds is entirely arbitrary, and ignores the possibility of a gradual change in type, from one extreme to the other, taking place in nature in response to alterations in environment. It has been shown elsewhere that there is good reason to conclude that such a change has actually occurred during the last 10 years in the polymorphic trypanosomes carried by wild *G. palpalis* on the Sese Islands (Duke, 1921).

(2) *The significance of virulence in the classification of trypanosomes.*

The time is at hand when we shall have to readjust our views on the relation of the mammalian trypanosomes to one another and to their hosts. Man's relation to these parasites is difficult to understand; but a solution to many problems will result from a proper realization of the true zoological affinities of the three trypanosomes at present known as *T. gambiense*, *T. brucei*, and *T. rhodesiense*.

Let us first endeavour to judge the case for the separation of *T. brucei* from *T. rhodesiense*, a separation based mainly on the behaviour of these organisms towards man. (We shall return later to a consideration of the alleged differences between *T. gambiense* and *T. brucei*.) A proper appreciation of the significance of virulence is essential to this inquiry, for much of the confusion at present existing in the classification of trypanosomes is due to the exaggerated importance that has been assigned to this character. The black man's position in relation to the game-tsetses, *G. morsitans* and *G. pallidipes*, also calls for careful investigation.

The virulence of any particular strain of trypanosomes is, essentially, a physiological character, and therefore should carry little or no weight in the differentiation of species.

Virulence varies greatly under different conditions of environment, and the factors which determine this variability are not yet properly understood.

The investigation of the virulence of a strain of trypanosomes involves a great deal of experimental manipulation, in the course of which the parasite is introduced into animals that it can never meet under natural conditions, and whose organization renders them entirely unsuited to the duties of host.

The ideal host for the mammalian trypanosomes is one in whose blood the parasite can exist for a long period, during which it has frequent access to its insect intermediary. Epidemic outbursts represent an unfavourable

crisis in the history of a trypanosome, and the conditions determining epidemic spread tend to render the parasite unsuited to cyclical development in the fly. A true understanding of the trypanosomes can only be reached by a judicious study of the parasites under natural conditions. The difficulties inherent in such a study compel us to resort to artificial laboratory tests, but we can and must discriminate in appraising our results. There are many obscure factors that come into play.

We know, for example, that the inoculation of trypanosome-containing blood into a susceptible animal is not invariably followed by the infection of that animal. Infection by the syringe is not a fair imitation of Nature's technique in direct transmission. In inoculation experiments we are ignoring altogether the cyclical method of infection, which, without a doubt, is Nature's orthodox procedure; and there are numerous instances where drastic modifications of virulence have arisen in the course of the prolonged upkeep of a strain under artificial laboratory conditions.

The so-called pathogenicity-test is intimately dependent on the conditions under which the experimental animal is maintained. As a rule nothing is said on this important point in the numerous publications about trypanosomes. In tropical laboratories, in particular, there is a tendency, after having performed the inoculation, to rely on the routine organization and the menial staff for the care of the animal until it dies. Where natives are concerned, this is not sufficient. Constant watch must be kept to see that every possible chance be given to the animal to fight its infection. Monkeys, in particular, require an immense amount of attention, and individual animals may vary greatly in their taste for food.

Often, too, persuasion is required to induce an animal to utilize the shelter of its box. With monkeys, and even more with dogs, exercise is essential to their well-being. Newcomers to the laboratory are unsuitable for experiment, until they have become acclimatized; and an animal in the early days of its captivity is depressed and miserable, and in no fit state to put up a fight against a trypanosome infection. In the case of monkeys, also, the different species vary greatly in their resistance to the parasites, and for comparative purposes the same species should always be used. When newly captured these animals are very liable to monkey-malaria, which, for a short time, may cause very pronounced febrile disturbance.

These points may seem irrelevant, but the advocates of the pathogenicity test should at least determine and record the virulence of their strains in terms of normal healthy animals.

There is a tendency at the present time to multiply the species of the parasitic protozoa, and no group of organisms has suffered more in this respect than the trypanosomes. It is difficult to defend the attitude of the species-mongers in regard to these parasites. Their system of classification rests on no sound zoological foundation, and merely tends to obscure still further a group whose real affinities we are but just beginning to appreciate.

Minor characters are arbitrarily selected as possessing specific value, and thereafter species spring up like mushrooms. It is surely a far sounder policy to search for fundamental affinities, than to exaggerate superficial and inconstant differences into characters of specific importance.

T. gambiense is said to be distinct from *T. brucei* on account of its chronic animal-reactions and the absence of posterior-nuclear forms. If a strain of *T. gambiense* acquires increased virulence during prolonged laboratory upkeep, it is quoted as an example of the effect of environment on virulence. Yakimoff's *T. gambiense* killed rats in 7–14 days, thus rivalling in fierceness the most virulent *brucei* strains, but it still retained its old name (Yakimoff, 1910).

Were such a strain recovered from game, no one would dream of calling the organism *T. gambiense*, even if posterior-nuclear forms were not at once discernible; yet at one stage in its career no doubt this ferocious organism was a quiet and unimpeachable *gambiense*, orthodox in all its reactions.

This is but one illustration of the unreliability of virulence, as manifested in laboratory animals, when applied to the classification of trypanosomes. And just as virulence is a physiological character liable to great variation in the individual strain, so the attitude of a strain towards the various mammals is a physiological character, and likewise subject to variation.

We know of instances, in the "proboscis-only" and the "proboscis-and-gut" groups of trypanosomes, of individual strains overcoming host prejudices that are sufficiently deep-rooted to influence the distribution of the organisms in nature. *T. vivax* has been found in man (Macfie, 1917), and in the course of laboratory upkeep this trypanosome has been persuaded to infect rabbits, which are unaffected by wild strains; similarly the alleged differences between *T. nanum* and *T. congolense* have been eliminated in the course of laboratory manipulations.

Why, therefore, should man be placed on a different plane from all the other food animals of the tsetse? We have an authentic instance of his infection by *T. vivax*, which has far less claim to the title of human parasite than has *T. brucei*. Does this human *vivax*-strain forfeit its right to its old name because of its detection in man? Unfortunately man is almost entirely outside the range of direct experiment, and our point of view with regard to him is apt to be biassed and old fashioned.

In classifying the big cats of Africa we should hesitate to sub-divide the lions into two species, those that do and those that do not eat man, because we know perfectly well that in certain circumstances any lion may become a man-eater. The parallel is not exact, but it serves our purpose.

The essential characters upon which the classification of the mammalian trypanosomes should be based are, first, the morphology of the organism; and second, its behaviour in the normal insect intermediary. *Identical morphology together with similar developmental behaviour in the fly should be regarded as proof of specific identity.*

(3) *Susceptibility to trypanosomes, and its relation to survival of a mammal in a fly-belt.*

Natives all over Africa avoid grazing their cattle in the bush in game-tsetse country. Their forefathers learnt wisdom by bitter experience, centuries before Bruce unravelled the connexion between the tsetse and the disease.

Numerous instances are recorded where the mere passage of susceptible animals through a fly-belt has resulted in their infection, and the trypanosomes in such case are undoubtedly conveyed by cyclically infected flies.

We are, indeed, forced to the conclusion that immunity or tolerance towards cyclically transmitted trypanosomes is an essential to the survival of a mammal in the fly-country. The susceptibility of domestic animals to trypanosomes renders their existence in the proximity of the fly extremely precarious, and cattle and dogs are rarely found in villages in the fly-country.

The Ndama race of cattle in West Africa is an exception to the general rule, and affords a striking illustration of successful adaptation to environment. These little animals are infected with trypanosomes, but, under good living conditions, they are not inconvenienced by the parasites; impairment of health, such as results from overwork and poor feeding, leads to a break-down of this resistance, and the animal succumbs to the trypanosomes (Roubaud, 1921). A similar instance of immune races of cattle occurs in the Lado (Austen, 1922). Ordinary domestic stock are susceptible to all the game trypanosomes, and simultaneous infection by two or more species must be very common.

Man is in a much more fortunate position. On a single occasion a *vivax*-like trypanosome has been seen in human blood, but no one familiar with the fly-belts of Africa will doubt that man, at the present day, is, for all practical purposes, absolutely immune to the "proboscis-only" and the "proboscis-and-gut" groups of trypanosomes. The rarity of human trypanosomiasis in the huge areas in which man and the game-tsetses come into contact, shows conclusively that he is also extremely resistant to *T. brucei*.

The odds against stock are thus vastly greater than those which confront man, and domestic animals pay the penalty for their susceptibility by succumbing to fly-disease in areas where man is unaffected.

There is good reason to believe that *T. vivax* and *T. uniforme* are relatively innocuous to well-nourished stock, and the same is true, to a less extent, of the more formidable *nanum-congolense* and polymorphic groups—provided that multiple infection of the same animal does not occur.

Stock infected experimentally with these trypanosomes and carefully fed and looked after can tolerate trypanosome-strains that prove rapidly fatal to poorly nourished animals. Unfortunately the climatic and other conditions prevailing in *morsitans* country are not as a rule conducive to good condition

in cattle, and, in the dry season at any rate, even healthy animals have great difficulty in picking up a living.

As far as we can tell, man, until recent times, has been unaffected by the trypanosomes of game-tsetse belts. But, as the Nyasaland Commission has pointed out, it is difficult or impossible to pronounce definitely upon this point (Bruce, 1915). It is only within comparatively recent years that the microscope has been employed in the diagnosis of native diseases, and it is possible that sporadic cases of human trypanosomiasis may have existed from time immemorial in inhabited game-tsetse areas.

The possible existence in man of a state of tolerance to *T. brucei*, similar to that shown by the game towards the same trypanosome, is difficult to prove. We look for enlarged glands, for the cord tied round the temples that tells of headache, for febrile and other symptoms, but it is doubtful if the ordinary routine methods of examining natives on a large scale, in *morsitans* areas, would detect infections too mild to cause symptoms. Moreover, when once a trypanosome is detected in the blood of a human being, treatment is commenced at the earliest possible moment. In the present state of our knowledge of the factors that govern virulence we dare not allow the disease to run its natural course.

The percentage of wild flies infected with the polymorphic group is such, in every game-tsetse belt hitherto investigated, that the inhabitants must constantly receive injections of salivary-gland trypanosomes. We know by experiment that some, at any rate, of these *brucei* trypanosomes at the present day cannot survive in man, whether they are injected by the syringe or by a naturally-infected fly. General observations in the field point to the same conclusion.

The French observers have adduced strong evidence that, in *palpalis* regions in the Congo, where human trypanosomiasis is endemic, the natives acquire an immunity which enables them to come to terms with the trypanosome. This contention, if established, places man in a relation to *T. gambiense* similar to that which the game occupies towards *T. brucei*; *i.e.* he is a warm-blooded host in which the parasite can lead a quiet life and provide for its posterity.

The conception of man as a mere mammal is essential to a proper understanding of the aetiology and spread of human trypanosomiasis. There is a general tendency, even to-day, to regard him as a comparatively recent victim to a group of parasites which, in the not very distant past, confined its attention to the beasts of the field. I have no doubt that for many centuries man has played a considerable part as a host, *in palpalis areas*, both to the polymorphic trypanosome and to the fly. This entails the existence of a state of equilibrium between man and the trypanosome in its cyclically transmitted form.

On the other hand, in *morsitans* and *pallidipes* areas, man has always been relatively refractory to trypanosomes carried cyclically by the fly. The same

strain when subjected to direct transmission may, however, behave in an entirely different manner; but we will postpone the consideration of this point until a later section.

(4) *The invasion of man by* T. brucei.

The opening up of tropical Africa, which has taken place in the last few decades, has had far-reaching effects. Traffic channels have been developed everywhere, along which pass, to and fro, men and beasts and their parasites. Communications now exist between areas formerly effectually isolated; and the equilibrium established in the course of ages between trypanosomes and their mammalian hosts, has been disturbed by the constant introduction of susceptible new-comers of every class.

Human settlement is incompatible with a large and unrestrained game fauna, and in former times primitive native settlement determined the range of the game.

The advance of civilization has dealt a heavy blow to the Big Game of Africa. In many parts of the continent, game has entirely disappeared from areas where it once existed in countless numbers. Almost everywhere its range has contracted, and its number diminished. This decrease in the game has reacted far more severely on the true game-tsetses, *G. morsitans* and *G. pallidipes*, than on *G. palpalis*, whose principal food animals are the black man, the hippopotamus, and reptiles, and whose habitat renders it but little dependent on ruminant game.

From the standpoint of the present discussion the most important effect on the tsetse of this disturbance in the game distribution is to compel the fly seriously to turn its attention to man as a food animal. There are many areas in Central Africa where native populations have lived for years in more or less close contact with one or other of the game-tsetses, without, as far as we know, suffering any inconvenience from trypanosomes.

Every Big Game hunter is familiar with the small villages scattered here and there in the fly-country—a few huts, and a patch of cultivation surrounded by a thorn hedge to keep off wild animals. The natives in such villages are constantly exposed to fly-bite. They possess no stock, or perhaps a few goats, and few or no dogs—ominous facts, testifying to the deadliness of the trypanosomes. These villagers rely to a certain extent on the game for their food. Fly inhabit the bush around the village, frequent the paths and waterholes, and may even be found in and about the huts. When the game becomes scarce the fly will naturally look to man to make good the deficiency. It may, in certain circumstances, follow the retreating game; but this is by no means an invariable occurrence, and the common result of scarcity of game is a concentration of the Glossinae along traffic routes and around the villages in the belt.

The change affects man in two ways: he is deprived of a source of food, and he receives the undivided attention of the fly. And so it happens that the

natives are subjected, with increasing frequency, to an ordeal which, under normal conditions, their forefathers withstood for ages—namely, the introduction into their blood of a copious dose of the salivary-gland forms of "*T. brucei*." Under such conditions the chances of the trypanosome obtaining a footing will be still further increased by any influence that debilitates the population, and I believe this to be the manner in which *T. brucei* establishes itself in man.

The origin of the recent outbreak in the Mwanza district can be explained on these lines. The spread of the disease is distinctly epidemic in type. At least 500 deaths have occurred in the course of some two years in an area where, it is estimated, 16,000 people are in possible contact with fly, though the actual bush-dwellers are much less numerous. In some villages over 20 per cent. of the inhabitants have died of the disease in 12 months, and in some of the smaller villages a higher percentage mortality has occurred.

There is no native history or German record of any similar disease having existed in the belt previous to this outbreak, but early in 1918 Dr Andrew Balfour saw a slide taken from a case of human trypanosomiasis in Mwanza hospital a short time before his visit. Dr Balfour, in reply to a query about this case, has very kindly sent me the following information: "The blood slide showed the heaviest trypanosome infection I have ever seen in a human being. The patient had died from what was originally thought to be cerebro-spinal fever." This patient came from Usmao, a fringe of which sultanate lies in the fly-country, so it is possible that the infection was acquired in this belt.

Late in 1918 or early in 1919 a case, the nature of which puzzled the local chiefs, was sent to Mwanza from a village in this fly area. The man eventually died in hospital, but no diagnosis was arrived at.

During 1916 and 1917 the whole Mwanza district was much disturbed by military operations, and the first recognition of the disease by the natives was associated with the appearance of a devastating famine in the belt, followed by a serious epidemic of influenza. About the same time the game, formerly very numerous in the belt, for various reasons diminished greatly in numbers.

The outbreak was thus immediately preceded by the very conditions already suggested as favouring the invasion of man by *T. brucei*. If my explanation of this outbreak is correct, the parasite first obtained a footing in debilitated man by the agency of cyclical transmission, but owed its rapid spread mainly, if not entirely, to the direct methods.

The appearance of the *rhodesiense* type of trypanosome near the southern shores of Lake Victoria dispels yet another illusion which has been held to support the non-identity of *T. brucei* with *T. rhodesiense*. Hitherto no case of *T. rhodesiense* in man has been recorded further north than lat. 9° south (Yorke, 1920).

The theory of origin just enunciated assumes that in certain circumstances man is infected in nature by flies cyclically carrying the *brucei* organism. What these circumstances are is as yet unknown, but I believe they have arisen

in comparatively recent times. We will consider this point again in section (5). The part played by ankylostomes, in preparing the human soil for *T. brucei*, calls for investigation. The Germans, in the days of the Uganda epidemic, found a large proportion of their Sleeping Sickness patients also suffering from ankylostomiasis, and the same holds good to a remarkable extent in the Mwanza outbreak. Bruce's Commission, in their account of the clinical manifestations of trypanosomiasis in Nyasaland, mention that local oedemas—especially oedema of the feet—were a common symptom (Bruce, 1915); and the most striking feature of the Mwanza disease, from the native point of view, was this very oedema of the dorsal surface of the feet. So constant indeed, was this oedema, that the natives based their diagnosis of the disease on this symptom. In view of the fact that a large proportion of the trypanosome-cases showed ankylostome ova in a single coverslip preparation of an uncentrifugalized stool, it seems unreasonable to ignore the possibility that the helminth was responsible for part of the syndrome. If this is so, the ankylostome infection may act as a powerful ally to the trypanosome in its endeavour to overcome the natural resistance of the native.

The case for regarding *T. rhodesiense* as an independent species rests on the weakest foundations. Up to the present, there is no evidence that a fly infected cyclically with *T. brucei* from game has ever infected a healthy human being, nor is there any practical justification for the common assumption that a fly cyclically infected with *T. rhodesiense* from man is henceforth infective to other human beings. Taute's big experiment does not justify the separation of these two trypanosomes. Taute (1919) was dealing with a single, or, at best, a limited number of strains of the parasite. These strains came from an area where there was no human trypanosomiasis; where, in other words, the fly and the trypanosome found their vital requirements satisfied by the game, and a serious appeal to man unnecessary. Taute's results throw light on but one phase of a complex problem, and show that, under normal conditions, man has nothing to fear from the polymorphic trypanosomes of a *morsitans* belt. The view here advanced is that the appearance of *T. brucei* in the blood of man is a departure from the normal course of events, and reveals Nature taken unawares by the general upheaval of the old order of things that has attended the awakening of a continent.

(5) *The spread of* T. brucei *in game and in man.*

There is no doubt that *T. brucei* relies on cyclical transmission by Glossinae for its propagation among the game.

The trypanosomes are not, as a rule, numerous in the peripheral blood of infected game-animals; indeed, they are more often demonstrable by sub-inoculation than by direct blood examination. The parasite, apparently, does little or no harm to its host, and in the blood of the antelope it is well adapted to cyclical development in the fly.

These considerations all point to cyclical rather than to direct transmission as the principal, if not the sole means of spread of *T. brucei* among ruminant hosts in nature.

Taute's experiment, referred to above, proves that, under certain conditions, *T. brucei* is incapable of survival in man. Similar results were obtained years ago on a very much smaller scale by Dr Ascenso (*Bulletin of the Sleeping Sickness Bureau*, vol. III. 1911, p. 174). In the majority of these experiments the trypanosome-containing blood was inoculated by the syringe into man; but it is, I think, fair to assume that the same results would have been obtained had cyclically infected flies been used. Taute, indeed, did perform an experiment with infected flies, and obtained a negative result.

So far as I know, no experimental inoculations of *T. brucei* into man have hitherto been carried out in an area where human trypanosomiasis of the *rhodesiense* type occurs, but I was fortunate, a few weeks ago, in obtaining evidence on this point.

Through the kindness of the authorities of Tanganyika Territory and Uganda, I was given the opportunity of visiting a "*morsitans*" belt[1] to the east of Mwanza, in which human trypanosomiasis has appeared in the last few years. A brief summary of the results obtained during this visit is given on page 259.

During the tour one of my fly-boys was bitten by a tsetse while he was sitting beside me cleaning slides in the porch of the tent. He caught the insect in the act of feeding on him, and I dissected it immediately. There was fresh blood in the anterior gut, and both the glands and the gut were swarming with trypanosomes. A few minutes before, I myself had been bitten by a fly which I just failed to catch. For various reasons I am certain that the insect caught on the boy was the same that had just bitten me. The camp was outside the fly-belt, and the only flies in the neighbourhood were some that had been caught on the previous day and kept alive in a cage, as time did not admit of their dissection overnight. Shortly before the biting incident, these flies had been killed, by liberating them one by one into a net and nipping the thorax of each insect. During this process one or two often escape, and I am sure that this is the explanation of the presence of the insect in the tent (cf. Duke, 1923).

I have had this boy and myself under close observation and daily blood examination ever since the episode, on 16 August, 1922. On 8 September, 1922, $1\frac{1}{2}$ c.c. of the boy's blood was added to citrate and injected into a monkey. Up to date (19 January, 1923) none of the three of us has shown any signs of disease.

This is yet another piece of evidence that the *brucei* organism, when carried cyclically by the fly, cannot infect man. The limitations of this experiment are obvious, but every scrap of evidence of this kind is of value.

[1] The fly implicated is not really *G. morsitans* but a closely allied species recently named *G. swynnertoni* by Austen (1923).

In this instance, moreover, the evidence acquires additional importance from the fact that the locality where these flies were caught had been specially selected as the most likely place in the whole belt to find flies cyclically infected with trypanosomes derived from man. Game had been scarce for the previous two months, and the fly were hungry and were feeding on man. In villages round about, recent cases of trypanosomiasis were detected.

Yorke (1920), discussing the significance of Taute's experiments, boldly stated his opinion that the same negative result would have followed had Taute used flies cyclically infected from a human case of *T. rhodesiense*. I think that Yorke was, substantially, right. But we must recognize the probable existence of a stage in a "*rhodesiense*" outbreak—*especially in the absence of game*—where certain strains may retain their power of survival in man even after cyclical passage through the tsetse. Different strains will differ in the extent to which they retain this power, and here the resistance of the individual host will no doubt exert an influence.

Yorke and Kinghorn and the Nyasaland workers believe that the great majority of the human race is naturally immune to *T. brucei*, but that a few unfortunate individuals in the community are devoid of this natural protection. These are they who develop trypanosomiasis, when bitten by a fly whose salivary glands contain *T. brucei*.

Now this hypothetic idiosyncrasy might either be constitutionally inherent in the individual or it might be induced by some depressing influence leading to a temporary or permanent reduction of his vitality. Taute has shown, however, that mere physical unfitness does not necessarily break down man's resistance to *T. brucei*. Presumably, therefore, this susceptibility must be a constitutional defect, which renders certain persons liable to infection by *T. brucei*.

I venture the opinion, however, that this explanation is inadequate. We know that at least 20 per cent. of the game carry *T. brucei*. If only young animals were examined, probably the percentage would be found to be very much greater. The game is infected, by the cyclical route, by the 0·2 per cent. of wild fly that carry the gut-and-gland trypanosome in nature. Admitting that the contact between man and fly is not, as a rule, so close as that between game and fly, it is none the less obvious that the inhabitants of a *morsitans* area must frequently be bitten by flies infective with *T. brucei*. Is it reasonable, therefore, to suppose that any person constitutionally susceptible to the ordinary *T. brucei* present in the game and fly around his village could reach puberty, much less advanced life, without succumbing to his handicap? Yet in Nyasaland the great majority of the cases are, I believe, in adults. A chronic disease, characterized by no outstanding symptoms, might easily escape notice in a native community, but a rapid illness such as that caused by "*T. rhodesiense*" would hardly be overlooked.

We must, I think, look for some other explanation of the occurrence of the disease in Nyasaland and Rhodesia. A negative result, in the circumstances

supposed by Yorke, need not necessarily be due to the natural immunity of the individual towards all strains of *T. brucei*; it would be equally explicable on the supposition that the power of survival in man was so feebly implanted in the particular strain of trypanosome under investigation that this power was eliminated during the cyclical development of the organism in the tsetse. In other words Taute's experimental natives, if bitten by a fly carrying another strain of *T. brucei*, might have become infected; whereas, if Yorke's explanation is correct, these particular persons are absolutely safe from all cyclically carried strains, be they *T. brucei* or *T. rhodesiense*.

It is possible, therefore, to explain the sporadic cases of human trypanosomiasis found in Nyasaland and Rhodesia by assuming that some of the wild-fly strains of *T. brucei* can infect man, while others—forming the great majority—cannot. No doubt the constitution and state of health of the individual will at times play a part in deciding whether or not he becomes infected. Ankylostomiasis may serve as an important ally to the trypanosome. The effect of the fly's food on the trypanosomes developing in its gut is not yet understood. Possibly the exposure of *T. brucei*, during its development in the fly, to a diet of human blood may modify the attitude of the parasite towards man, just as the normal inability of a bacillus to ferment certain sugars may be overcome by repeated culture of the organism on a medium containing that sugar. The German workers are convinced that it is the character of the trypanosome that determines the infection of man, and not the resistance of the individual human host. According to Taute's view, *T. rhodesiense* will always infect man, and *T. brucei* will never do so. The Germans, however, go too far when they assign to the behaviour of the parasite in man's blood the importance of a specific character.

The true explanation lies, I believe, between these two extremes. *T. brucei* belongs to a group of trypanosomes one member of which is an acknowledged parasite of man. Under primitive conditions in game-tsetse areas *T. brucei* loses touch with man, and man must be regarded as very refractory if not immune to this trypanosome, as are baboons at the present day. There is no evidence that he is tolerant, as is the game; but he is obviously not highly susceptible, as are his domestic animals.

The danger to man lies in the fact that *T. brucei* requires relatively little inducement to overcome its disability to utilize man as a host. In Nyasaland and Rhodesia there are, apparently, human strains of *T. brucei* carried by wild *G. morsitans*. How these strains acquired the power to infect man it is impossible to say, but I believe that they are of relatively recent appearance, and that the state of affairs in these two areas does not represent the normal relation of *T. brucei* to healthy man.

Turning now to *T. rhodesiense* in epidemic form, the only instance of this hitherto recorded is the outbreak near Mwanza, to which reference has already been made. Here the history and the distribution of the disease point to direct transmission as the principal means by which the trypanosome passed from

man to man. An extract from Swynnerton's Report (1923, p. 338) will best serve to illustrate the extraordinary degree of contact attained between fly and man in this fly-belt.

Describing the type of village classified as "completely in the bush," he writes:

A small triangle formed by native paths through high weeds. At each of two of its corners a hut, at the third two. A diminutive and dirty open space before each hut and a shade-tree at two of them. The general thorn forest... extending right up to two sides of the triangle. The third side abutting on a limited open space of grass and weeds representing old cultivation, itself surrounded by the bush. A path leading through this and then through mgongwa-with-thicket wooding, past a granite kopje, in which we found puparia, to a water-hole in the bush. Another bush-path leading to the main piece of cultivation some distance off in the bush in another direction. Fly fairly abundant in the thorn-bush near, but less so than in the village. Here it seemed concentrated and greatly at home. An old woman who appeared was accompanied in all her movements and domestic duties by tsetses of which she took little notice. A dozen or more at a time would be perched on her leather garments and her skin. A number followed her into her hut and remained there. One of my fly-boys went in and captured six. About and between the huts we were much attended, and a few fed successfully...and from the very late moment at which I ordered a separate catch, over 70 were taken.

One of the solitary huts was deserted. The woman had died of sleeping sickness, and her husband, the headman, had gone; apparently it was not known whither, or whether he was infected. Of the four remaining inhabitants one man still well was away accompanying one of the others, a young man with sleeping sickness, to...hospital; the old woman has been mentioned, and a younger woman "had been ill with *malali*[1] but was now better, and had gone some days before to visit in Igombe." I found her bed had just been slept in—a probable case of concealment.

It may be objected that this intense fly infestation is equally propitious to cyclical transmission. This is to a certain extent true, and constitutes one of many difficulties in deciding between the two methods in nature.

In the case of the Mwanza outbreak, however, certain important facts point to direct transmission as the predominating factor in the spread of the trypanosome. The manner in which the disease spread from village to village in the wake of an infected visitor is very striking; and, in some instances, the interval which elapsed between the appearance of the first case in a village and the recognition by the natives of symptoms in the second case was so short that cyclical transmission could hardly have functioned.

Another important point is that the percentage of wild flies carrying the gut-and-gland trypanosomes in the Mwanza area, as revealed by dissection, is approximately the same as that obtaining in the Masindi *morsitans* belt in Uganda (where there has never been any human trypanosomiasis), and in Nyasaland (where the number of cases is very small).

[1] *Malali* is the native name for human trypanosomiasis in this area.

Bruce's Commission in 1912 found 0·10 per cent. of flies carrying the gut-and-gland trypanosome in a total of 1975 dissected; in 1913 the percentage found was 0·09 in 1060 flies (Bruce, 1915). The corresponding figure obtained by Miss Robertson and myself for the Masindi area was 0·19 per cent. in 1562 flies (Duke, 1916). The Mwanza figure for the gut-and-gland group is 0·13 per cent. in a total of 2206 flies dissected (Duke, 1923).

In parts of the Mwanza fly-belt where game is comparatively rare and the fly is, consequently, more dependent on man for food, the percentage of gut-and-gland infections is less than the average for the belt as a whole. I visited one such area where, for two months previous to my visit, game had been rare, and found that the "*game-index*" of the fly—that is to say, the percentage of flies showing proboscis-and-gut and proboscis-only infections—was 4·1, as contrasted with an index of 9·8 for an area where game and fly were in closest contact, and of 8·4 for the belt as a whole. The population of the area numbered about 440, and of these about 30 per cent. appeared for examination. 21 per cent. of these had large axillary glands—in some cases the glands measured $1\frac{1}{2}$ inches in one diameter—and in three cases trypanosomes were found in the first 20 fields of the slide of peripheral blood examined. Several deaths from trypanosomiasis had occurred in these villages within the month preceding my visit.

The infectivity figure for the gut-and-gland group in this area was 0·10 per cent. (722 flies dissected), as contrasted with 0·24 per cent. for the game-and-fly area (819 dissected) and 0·13 per cent. for the belt as a whole (2206 dissected). It is, however, worthy of note that the total for the whole belt includes 665 dissections performed in an uninhabited game-and-fly area; no gut-and-gland infections were found in these flies, although *T. brucei* was recovered from the blood of the game of this area.

It may be retorted that the existence, in this relatively gameless area, of flies with gut-and-gland infections, is evidence that these trypanosomes are derived from human beings, and that the disease is therefore spread by cyclically infected flies. That some, at any rate, of the human strains in the Mwanza infected area are capable of cyclical development in tsetse has already been demonstrated by experiments which are still in progress at Entebbe. The two human strains brought back for investigation both develop readily in laboratory-bred *G. palpalis*.

But, apart from the possibility that the game is responsible for the gut-and-gland fly infections in this area, we have to face the significant fact that the only gut-and-gland fly caught in this area bit a European and a Muganda native without infecting either with the trypanosomes with which its salivary glands were swarming.

As is the case elsewhere throughout the infected area, the majority of the cases in the locality under discussion were adults or old people; and it is difficult to believe that the explanation of the distribution of the disease in this locality lies in individual idiosyncrasy towards a trypanosome to which

the great majority of the population is immune. Three months before my visit both game and fly had been much more common around these villages; and before the general diminution throughout the belt, game was present in large numbers in this locality. It seems hardly conceivable that anyone handicapped by a hereditary susceptibility to game trypanosomes could have lived long enough in this area to reach adolescence.

The information yielded by these fly dissections—small though the totals are—is, none the less, of considerable interest.

We can safely assume that *T. brucei* was present in the game and fly of the Mwanza belt before the appearance of the human disease. During my tour, the *brucei* type of organism was recovered from game, and the biting incident just described shows that the wild fly also carried this trypanosome. There is no reason to doubt that the game of the belt is as heavily infected with *T. brucei* as it is in *morsitans* belts. The agreement shown by the percentage figures for the gut-and-gland organisms in the various "*morsitans*" areas under review, suggests very strongly that 0·2 per cent. is about the maximum carrying capacity of wild *G. morsitans* and *G. swynnertoni* for this group of organisms.

In the Mwanza area a brisk outburst of human trypanosomiasis has occurred, caused by a member of the gut-and-gland group, but there is no corresponding increase in the percentage of flies cyclically infected with this group of trypanosomes. I am convinced that the disease in this fly-belt is of recent appearance and has not existed, in its present form at any rate, for more than a few years. The fly are now undoubtedly feeding on man. Either, therefore, the new disease is spread by some other means than by cyclical transmission, or the normal *T. brucei* in the belt has suddenly become pathogenic to man. We have seen that the available evidence favours the former interpretation.

The evidence derived from the fly-dissections, therefore, supports the view that the human disease in the Mwanza area is spread by direct as opposed to cyclical transmission; and, incidentally, it adds yet further weight to the contention that *T. brucei* and *T. rhodesiense* are one and the same species.

The character of the *rhodesiense* disease in man favours direct transmission. We have seen in Part I that increased virulence, acquired in the course of prolonged upkeep by direct transmission, may eventually be accompanied by the loss of the power to develop in the fly. In other words, when the epidemic has reduced the population to the limit below which direct transmission cannot function on a large scale, the "outbreak of human trypanosomiasis" dies out.

When an epidemic works itself out, as it certainly will, what happens to the trypanosome?

During the earlier stages of an epidemic the parasite must frequently be exposed to chances of cyclical development in the fly, and doubtless most of these early strains are successful. The probable result of cyclical passage through the insect at this stage will be the loss of the newly acquired power

of survival in man. But, as the epidemic progresses, and especially if the disease is not very virulent, it is possible that this power to survive in man may become so firmly rooted in the strain that it can survive the test of cyclical transmission by the tsetse. In this way a cyclically transmitted human strain may be established in wild fly.

The future career of the trypanosome as a human parasite probably depends on the extent to which game is available to the fly. This point will be dealt with in section (9).

Apparently direct transmission plays a much more prominent part in the spread of trypanosomes through human communities than it does among the game. It is well known that the proboscis-and-gut group utilizes this method of spread to a considerable extent, and many of the most virulent strains of *T. congolense* seem to rely entirely on mechanical transmission.

There is no evidence that the gut-and-gland group uses this means of spread save in the case of man, though it is quite possible that virulent strains may in this way be developed in domestic animals. There is, so far as I know, no instance of any trypanosome becoming virulent to game in nature; but it would be extremely difficult to demonstrate the existence of such a phenomenon, if it did occur.

The importance to the gut-and-gland group of trypanosomes of cyclical development in the fly cannot be denied; it is the normal means by which these organisms are spread in nature, and are purified from disadvantageous variations such as enhanced virulence. Direct transmission, on the other hand, defeats the objects of the parasite by the rapid destruction of its host; and we have seen that strains so transmitted may eventually lose altogether the power of survival in the fly.

I believe that wherever human trypanosomiasis exists in epidemic form the method of transmission will be found to be direct.

(6) *The proboscis-and-gut group of trypanosomes and its analogy to the gut-and-gland group.*

In the preceding sections we have attempted to show that the barrier erected between *T. brucei* and *T. rhodesiense* is both artificial and arbitrary, and that there is no sound zoological reason for separating them.

In the course of the discussion we have inevitably touched upon the relation of *T. brucei* to *T. gambiense*, and thus to some extent anticipated what properly belongs to section (8). But before leaving the *rhodesiense-brucei* debate we will turn for a moment to the proboscis-and-gut group of trypanosomes, the study of which lends striking support to the views put forward above about these two parasites.

The parallel between the two groups is remarkably close; the various strains of the proboscis-and-gut group are indistinguishable morphologically, and their behaviour in the fly is the same.

As in the gut-and-salivary-gland group, several species are described on the strength of differences in virulence.

The *nanum-congolense* trypanosomes can be spread in nature by direct transmission by biting flies other than the Glossinae, and directly-transmitted strains are often characterized by a relatively high degree of virulence, particularly to stock.

No member of the group is known to infect man. But whereas among the mammalian hosts of the gut-and-gland trypanosomes, man is distinguished by his immunity to certain of the so-called species, in the proboscis-and-gut group the dividing line runs between ruminants on the one hand and animals such as dogs, hyaenas, and monkeys on the other. The strains that cannot survive in carnivora and in monkeys are known as *T. nanum*; those that can, as *T. congolense*.

Now in studying this group we can resort to direct experiment in examining the constancy and significance of the alleged differences, and the information so obtained can be applied to the less accessible polymorphic group.

By feeding small batches of wild flies on monkeys and then dissecting all the insects, a very exact supervision can be kept on the infectivity of the various strains. In this way I have proved the presence of both the *nanum* and the *congolense* forms in wild *G. morsitans* in Uganda; and I have found that some of these wild-fly strains can infect dogs and monkeys, some cannot (Duke, 1916).

It is possible, from Bruce's results in Nyasaland and my experience in the Mwanza area, that monkeys are less susceptible than dogs to *T. congolense*.

An interesting result was obtained during the Mwanza tour. A batch of 80 wild *G. morsitans* was fed on a clean healthy dog. Dissection of the flies immediately after feeding revealed two with proboscis-and-gut infections, and both of these had fed on the dog. On the 10th and 11th days after the feeding, trypanosomes were found in the dog's blood. From then onwards the animal was negative to daily thick-film examination until the 34th day, since when it has shown parasites in very small numbers from time to time. At the time of writing this dog appears to be in excellent health, 170 days after the original infection.

T. congolense (= *pecorum*) usually kills dogs rapidly, and this wild-fly strain illustrates very well a degree of virulence midway between that of *T. nanum* and *T. congolense*. Bruce's Commission also records an instance of a dog recovering from an infection with this trypanosome (Bruce, 1914).

Organisms corresponding to *T. nanum* and *T. congolense*, respectively, were obtained from game and fly by Bruce in Nyasaland and by Kinghorn and Yorke in Rhodesia, so that it is probable that the two forms of trypanosome occur wherever the proboscis-and-gut group is found.

T. nanum is probably everywhere the more common form; and Mr Richardson, Veterinary Pathologist, Uganda, has confirmed my impression that this

type is, as a rule, less virulent for stock than *T. congolense* (= *pecorum*). It is important to note that both of these trypanosomes are present in cyclically infected wild flies.

In the case of *T. brucei* and *T. rhodesiense*, which are also separated from one another because of their host preferences, there is, as yet, no definite proof that the latter occurs in wild flies; at all events *T. brucei* is by far the more common in nature. When, however, *T. nanum* is subjected to the artificial conditions of the laboratory, we find that the difference between it and the *congolense* type vanishes. Yorke and others have persuaded *T. nanum* to infect the small laboratory animals, to which it is, in nature, non-pathogenic.

At Mpumu the strain of *T. congolense* (= *pecorum*), which was kept up in monkeys, after a time showed signs of losing its virulence towards these animals. In the course of attempts to transmit this strain cyclically by laboratory-bred *G. palpalis*, it was found that flies heavily infected in both proboscis and gut failed to infect clean monkeys (Duke, 1912).

Looking back on these experiments, I think it probable that the failure was due to the strain having lost its pathogenicity for monkeys during the passage through the fly; had a ruminant been employed as the test animal, probably infection would have resulted. In other words the fly took up *T. congolense*, and turned out *T. nanum*.

It is evident, therefore, that the alleged differences between these two trypanosomes are merely variations of a physiological character, and should not be utilized in classification.

The evidence supplied by the proboscis-and-gut group supports the opinion advanced in section (2) on the inadequacy of this character as a reason for separating *T. brucei* and *T. rhodesiense*.

If in place of man we substitute the laboratory animals, there is a close analogy between the two groups of trypanosomes. There is no reason to doubt that Yorke's experiments with *T. vivax* and the small laboratory animals, repeated under similar conditions with *T. brucei* and man, would yield analogous results; indeed, Lanfranchi's infection with a strain of *T. evansi* is most probably a case in point (Mesnil and Blanchard, 1914).

(7) *Man's relation to the polymorphic trypanosomes of* palpalis *regions. Direct versus cyclical transmission.*

We have already discussed man's relation to the fly and its trypanosomes in game-tsetse regions. It remains to investigate his rôle as a host in *palpalis* areas.

Here the problem is more complicated, because man, as one of the principal mammalian hosts of the fly, has been, for ages, intimately associated with its trypanosomes. On biological grounds, therefore, we should expect to find the native in *palpalis* regions biologically adapted to the polymorphic trypanosomes carried by this fly, in the same way that the game is adapted to the organisms carried by *G. pallidipes* and *G. morsitans*.

Now it is a remarkable fact that wherever man and *G. palpalis* have been in age-long contact, human trypanosomiasis is found in some form or other. There are exceptions to this generalization, but their rarity merely emphasizes the truth of the assertion.

We have already commented on the difficulties attendant on the study, by man, of trypanosomiasis in man. The conditions of life in native communities are very different from those obtaining among even the most gregarious of game-animals. More or less permanent residence in a limited area, the differentiation of labour existing even in the most primitive human society, the proximity of domestic animals, the liability to influences leading to wholesale reduction in vitality—these are all factors which tend to confuse the student of the biology of the parasite.

Again, the value attaching to a human life, however humble, focusses attention rather on the relief of the individual than on the search for the causes that determine the spread and virulence of the trypanosome in the community.

The epidemiological studies of the French workers in the Congo have brought to light some very valuable information. They recognize two types of the disease in the *palpalis* areas of this part of Africa. There is a mild endemic form of human trypanosomiasis, which has been known for years to the inhabitants; and there is also an epidemic form, which is much more virulent and which causes, at times, an appalling mortality. Epidemic outbursts may occur in an endemic area, and after such an outburst the disease may once again assume its milder endemic form.

Correlated with the existence of these two types, there are differences in the age-incidence of the disease in the community. Where it is endemic, the young of both sexes show the greatest proportion of the infects and suspects. In epidemic areas, on the other hand, young and old are attacked equally. Thus, writing of the Haute Likouala district in the French Congo, Clapier (1921) remarks: "La trypanosomiase est dans ce petit groupe très intense à la fois chez les adultes et chez les enfants, ce qui semble bien indiquer une poussée épidémique récente."

According to the French observers, this difference indicates that in the endemic areas almost everybody is infected in the days of his youth, but that, with increasing years, a labile immunity is acquired that protects him from any further inconvenience. In its epidemic form the disease attacks old and young alike, and there is, at first at any rate, no immunity and, in consequence, a very high death rate.

Discussing the two types of the disease in the district of the Congo, Jamot (1920) writes: "Dans les zones où la maladie est endémique, nous n'avons enregistré pour 518 malades, que 132 décès, soit une mortalité de 25 %; par contre, dans celles où elle sévit sous la forme épidémique, nous avons compté, pour 1919 malades, 1349 décès, soit une mortalité de 70 %."

Certain French workers have long insisted that mosquitos play an important part in the spread of human trypanosomiasis in the Congo. Experimentally

these insects have been shown to transmit trypanosomes by the direct method (Heckenroth, 1913). No one who has heard the roaring swarms of *Mansonia* and other culicines, so often present in native villages near the water, can fail to be impressed by the possibility of the insects aiding in the spread of human trypanosomiasis—provided that the parasites are sufficiently numerous in the peripheral blood. But it is by no means certain that any other insects than the Glossinae do actually function on a large scale in the spread of human trypanosomiasis in nature. My own observations have convinced me that tsetses are much more prone to flit from host to host in their efforts to feed than are mosquitos, and the structure of their proboscis parts makes them more suited to direct transmission.

Bagshawe points out that the evidence afforded by the Uganda epidemic tells against mosquito agency; but a tsetse may well succeed where a mosquito will fail. My experience, over several years, of the collection of monkeys at the Entebbe Laboratory confirms the inadequacy of the mosquito as a natural transmitter of trypanosomes. At times there are as many as 60 monkeys living at the laboratory, the boxes being just out of chain-reach of one another. Instances have occurred where a baby monkey has been allowed to run free among the others, the majority of which are infected, including its own mother, in whose box it spends most of its time. No case of casual infection has occurred, although one of the strains maintained at the time was the virulent direct-transmission strain referred to in Part I. Culicine mosquitos are numerous, and malarial infections very common among the monkeys.

In this connexion Clapier (1921) makes an observation that is very significant. He writes: "Le parasite se retrouve donc très souvent dans le sang au simple examen direct *dans les pays où la maladie est à l'état épidémique.*" It is plain, therefore, that the disease in its epidemic form in these regions presents one of the features that are essential to direct transmission, *i.e.* large numbers of trypanosomes in the peripheral blood.

Jamot (1920) has collected interesting statistics indicating that direct transmission, by some insect, must play a part in the spread of the disease in epidemic form. In an epidemic area in which 5347 cases were detected, he found 954 instances where two individuals of the same family were infected, 253 of three in a family, 74 of four, 24 of five, and one instance in which 17 persons of one family were all infected. Evidence such as this speaks irresistibly in favour of direct transmission.

In human communities in *palpalis* regions we must, therefore, recognize the existence of *two types of trypanosomiasis due to the gut-and-gland group: a mild cyclically-transmitted endemic form, and a virulent epidemic type that relies mainly on direct transmission for its spread.*

The endemic strain is not necessarily a menace to the community. Under good living conditions this strain may cause man but little inconvenience, but ankylostomiasis and other debilitating influences will doubtless exert an unfavourable influence on the course of the disease in the individual.

If the population of a *palpalis* area increases beyond the limits of safety, and, in consequence, *the contact between fly and man becomes sufficiently intense*, then direct transmission may intervene and an epidemic burst out. Any cause that weakens the general vitality of the community will tend in this direction by releasing the parasite from the restraint of its host's normal resistance, and favouring rapid multiplication in the peripheral blood.

Once a strain has acquired the degree of virulence essential to rapid propagation by direct transmission by Glossinae, it is in a favourable position to avail itself of other biting flies, such as mosquitos.

Epidemics of trypanosomiasis in *palpalis* areas tend to die a natural death. They usually leave behind them an endemic form of the disease, established in the fly—a state of affairs very similar to that existing before the epidemic arose. The future of these endemic strains will depend on the conditions that result from the epidemic. If man remains the principal mammalian host of the fly, then we shall see the gradual establishment of an equilibrium, more or less exact, between him and the trypanosome. If game animals replace man, the history of Sese will, I believe, be repeated: the trypanosome, after cyclical passage for a number of generations in ruminants, will lose its power of survival in man. The length of time needed for this transformation will depend, presumably, on the degree to which the power of survival in man is engrained in the constitution of the trypanosome.

(8) T. brucei *versus* T. gambiense.

In the literature of trypanosomiasis these are two firmly established species. A number of tests have been devised by which they can be distinguished; indeed, so much stress has been laid on their points of difference, that the much more striking and significant resemblances have, to a great extent, been overlooked.

Until recent years, *T. gambiense* occupied a unique position among trypanosomes by virtue of its power of parasitizing man, and the recognition accorded to *T. rhodesiense* as a separate species tended still further to isolate the earlier known human trypanosome.

Speaking of the Nyasaland investigations Bruce (1915) says "the Commission came to the conclusion that...*T. gambiense* and *T. rhodesiense* were different species," and this pronouncement therefore has great authority behind it. Bruce and his co-workers hold, however, that *T. rhodesiense* and *T. brucei* are the same species.

The main points by which *T. gambiense* and *T. brucei* are distinguished are:

(1) Behaviour with human serum.
(2) Animal reactions—chronic or acute.
(3) Cross-immunity reactions.
(4) Pathogenicity to man.
(5) Presence or absence of posterior-nuclear forms.

A study of the literature reveals that all these distinctions have been proved inconstant save the last two, and we have seen that no specific value can be allowed to either of these.

According to the present arbitrary system of classification, no trypanosome which can, under any circumstances, develop posterior-nuclear forms, can bear the name of *T. gambiense*. In much the same way, *T. brucei* is debarred from all dealings with man under pain of loss of its title.

The other tests have all given way. Thus there are numerous instances of *T. gambiense* developing intense virulence in laboratory animals, in which, when freshly isolated, it causes only a mild disease. Mesnil (1912) has recorded identical eye-symptoms in fowls inoculated with *T. gambiense* and *T. rhodesiense* respectively. Bevan (1912) reported opacity of the cornea in a sheep inoculated with *T. gambiense*—a symptom usually associated with *T. brucei*: while Laveran (1912) finds that *T. rhodesiense* soon loses its susceptibility to human serum, and Mesnil and Ringenbach have observed a protective and curative action of human serum on *T. gambiense* (Mesnil, 1912). Zeiss (1920) describes similar variations.

A change from the *gambiense* to the *brucei* form of trypanosome has apparently taken place during the last 10 years on Sese (Duke, 1921); and as regards the alleged differences between *T. brucei* and *T. rhodesiense* we can at least claim that the case is unproven.

T. gambiense and *T. brucei* manifest identical morphology in man and in monkeys and in stock, and they behave in the same way in the insect intermediary. Their distribution coincides roughly with that of *G. palpalis* in the one case and the game-tsetses in the other, and their principal mammalian hosts are, respectively, man and game. Differences in environment are amply sufficient to account for such differences as exist between the two trypanosomes.

I feel confident that epidemic strains of *T. gambiense* will be found in *palpalis* areas in the Congo, and that these, under suitable handling, will yield posterior-nuclear forms.

Meantime, why should not the onus of proving that *T. brucei*, *T. gambiense*, and *T. rhodesiense* are separate species, lie with those who hold this view?

(9) *The Big Game as a reservoir for trypanosomes, especially for those of man.*

Of recent years there has been a great deal of discussion about the African Big Game as a reservoir for the trypanosomes that affect man. The game, in nature, is closely associated with the Glossinae, and, in consequence, with the trypanosomes carried by these insects. With the exception of *G. palpalis*, whose principal food animals are man and reptiles, the various species of Glossinae rely chiefly on the game for their food, and a state of equilibrium exists between the ruminants upon which the fly feeds and the trypanosomes that it carries.

The Big Game is, undoubtedly, the primitive and principal reservoir of the three groups of mammalian trypanosomes that rely on tsetse for their cyclical transmission. In these three groups fall most of the so-called pathogenic species, exceptions being *T. evansi*, *T. equiperdum*, and *T. equinum*, of whose genesis and affinities we know little or nothing.

Man and his domestic animals occupy a peculiar position among African mammals in relation both to the fly and to its trypanosomes. Domestic animals differ from their wild relatives in possessing comparatively little resistance to trypanosomes. The Ndama race of cattle has been mentioned as an exception to this generalization, and there is no doubt that the resistance of stock to the pathogenic trypanosomes varies in different areas in response to different environmental conditions. Roubaud (1921) has called attention to the influence of "bien-être" on the struggle of a mammal against haematozoan parasites, and this factor certainly plays a very important part in the survival of stock throughout Africa. According to native report, cattle on the Islands of Victoria Nyanza flourished exceedingly in the days before the great epidemic of human trypanosomiasis in Uganda; and there is no reason to doubt that at any rate the proboscis-only and the gut-and-gland groups of trypanosomes were present in the wild-fly of that period. No doubt the splendid grazing afforded by these fertile islands enabled the animals to tolerate their parasites. But in spite of these exceptions, man's domestic animals cannot be regarded as natural hosts of the pathogenic trypanosomes.

We now come to man himself, in whom all our interest centres. In *palpalis* areas the tsetse draws heavily on man for food. He is, in consequence, constantly being inoculated with the parasites carried by the fly. As far as is known, man is absolutely immune to the proboscis-and-gut trypanosomes, and only a single instance is recorded of his infection with a member of the proboscis-only group. On the other hand, a member of the gut-and-gland group of trypanosomes—*T. gambiense*—has long been known as a human parasite; and the experience of the French in the Congo indicates that a balance may be established between *T. gambiense* and man, though it is less perfect than that existing between *T. brucei* and the game. Thus, in a fly-area where man is the favourite mammalian host of the tsetse, we find him biologically adjusted to serve as host to the gut-and-gland trypanosome carried by that tsetse.

Turning now to the game-tsetse regions, we find that the relation of the game to man is more complicated. Under primitive conditions man does not figure prominently in the dietary of the game-tsetses. The fly will feed upon him readily enough, but, until recent years, he has played a negligible part as a food animal in comparison with the game. As far as we know, in game-tsetse regions man's immunity to all three groups of tsetse-carried trypanosomes has, until recently, been complete; if mild forms of human trypanosomiasis existed in the past, they were not sufficiently pronounced to leave any trace in local native tradition. But in recent years a member of the gut-and-

gland group has appeared as a human parasite in some of the game-tsetse regions of Southern Africa. These human strains are indistinguishable from trypanosomes of the same group occurring in the local game.

The percentage of wild flies capable of cyclically carrying the gut-and-gland trypanosomes appears to be about 0·2, and this percentage is responsible for the infection of at least 16 to 20 per cent. of the local game.

Except in the Mwanza area, however, the percentage of infected individuals, in the populations exposed to the fly, is very small, and the discrepancy is not entirely explicable by the greater exposure of the game to the tsetse. Man undoubtedly possesses a high degree of immunity towards the gut-and-gland trypanosomes of game-tsetse areas.

Now for many years previous to the first recognition of human trypanosomiasis in the tsetse areas of Southern Africa, the spread of civilization had been opening up the country, disturbing primitive relationships, and exposing the natives to new influences. As man pushed back the game, he unwittingly filled the gap which his advance had caused in the dietary of the fly, until, in some areas, the reclamation he achieved was sufficiently thorough to deprive the insect of the shelter necessary to its existence.

It is, I believe, during the period when the game of a district is disappearing and the tsetse are looking round for a substitute, that the native's natural immunity to trypanosomes is most likely to break down.

The history of the outbreak of human trypanosomiasis in the Mwanza belt seems to be an example of this process. The fly in this area show a most unusual partiality for man. Swynnerton, in his Report of the epidemic to the local authorities, writes that "a striking point about this fly is that it shows no appreciable preference for cattle as against man"; and he states that his fly-boys even considered that the fly preferred man to cattle. Native reports were unanimous that the game had greatly diminished during the last few years. The important point is that the fly has, apparently, resorted to man in consequence of the relatively sudden failure of the supply of game blood, and this suggests the first tentative conclusion from our inquiry:

The game constitutes an easily accessible source of food with which the fly can satisfy its requirements without recourse to man. The risk of the invasion of man by the trypanosomes (which are carried by the insect, and which are potentially able to infect human beings) *thus diminishes in proportion to the number and accessibility of the game.*

But there is another equally important aspect of the question. Man in *palpalis* regions is a regular host to a member of the gut-and-gland group—*T. gambiense*. In game-tsetse regions, on the other hand, human trypanosomiasis is rare, although 16 to 20 per cent. of the game is infected with *T. brucei*.

According to the view advocated here and elsewhere (Duke, 1921), *T. brucei* and *T. gambiense* are variants of the same species. Comparing the conditions prevalent in *palpalis* and in game-tsetse areas respectively, we find that a tsetse and a gut-and-gland trypanosome are common to both, but that the

principal mammalian host of fly and trypanosome is different in the two cases —in the former, man, in the latter the game. That the differences between the *gambiense* and *brucei* forms of the trypanosome are not due to differences in the habits and habitat of the Glossinae concerned with their transmission, is shown by the existence, in the wild *G. palpalis* of the Victoria Nyanza, of a trypanosome answering in all essentials to *T. brucei*, and by the fact that on certain of the depopulated islands of this lake at the present day, *G. palpalis* is feeding most freely on antelope.

I have suggested that the form assumed by the trypanosome is determined by the class of mammal in which it spends most of its time—man in the one case, ruminant game in the other. The Sese Islands of Victoria Nyanza afford valuable confirmation to this view. It may be objected that *T. brucei* has been introduced into the islands since the depopulation. But Damba Island, where most of the observations were recorded, is effectually isolated from all outside sources of *T. brucei*; and the fact remains that nowadays the *gambiense* form of trypanosome has apparently disappeared from the wild fly of the north-western lake.

We must, therefore, recognize the possibility that *the game plays a very important part in determining the behaviour of the gut-and-gland trypanosome towards man.*

Current views on the African Big Game as a trypanosome-reservoir require revision. There is no doubt that the game is the principal reservoir of the Glossina-carried trypanosomes that attack man's domestic animals. With the human trypanosomes, however, the case is different. The human strains can all thrive in game animals, but there is a considerable amount of evidence to show that sojourn in wild ruminants leads to the assumption by the gut-and-gland trypanosomes of a form in which they cannot infect normal healthy man.

We must also credit the process of cyclical development in the tsetse with a share in the disarming of the trypanosome. Man is absolutely refractory to many animal diseases, among which are East Coast Fever and Rinderpest, and we may safely regard him as possessed of a powerful generic resistance to trypanosomes. But this resistance is not complete. It was broken down centuries ago in *palpalis* areas, and it is giving way under our eyes in response to the massed attacks of *T. brucei* which modern conditions have made possible in some game-tsetse areas.

Cyclical transmission preserves deep-rooted characters; and, in the course of time, as man is constrained to co-operate with fly and trypanosome, the power of the parasite to utilize man may survive this ordeal. But the process is reversible, and with the reappearance of naturally tolerant ruminant hosts, man may recover his lost prestige.

To sum up: The game, in game-tsetse areas, is the main natural reservoir of the proboscis-and-gut and proboscis-only groups of trypanosomes; but inasmuch as we must assume that man, under exceptional circumstances, can be infected by wild flies cyclically carrying the gland-and-gut group, the game

is also *a* reservoir, in game-tsetse areas, of the human parasite known as "*T. rhodesiense.*"

In *palpalis* areas man is the main reservoir for the trypanosome known as *T. gambiense.* In game-tsetse regions man is probably a more important reservoir than the game for human strains of this group of trypanosomes, because direct transmission (from sporadic human cases infected by the cyclical route) may cause an epidemic outburst in a human community. It is also possible that there is, in the process of adaptation to man of the *brucei* form of trypanosome, a stage at which cyclical transmission from man to man occurs.

But in both types of fly area, and particularly in game-tsetse areas, the game acts as a shock-absorber in regard to man; and this beneficial protective action probably considerably outweighs its power for harm as a reservoir. The presence of the game simultaneously diverts the attention of the tsetse from man, and leads to the suppression in the trypanosome of the potential qualtity of survival in man.

The practical outcome of these conclusions is that *we should concentrate our attack directly on the fly,* which we know to be an indispensable factor in the spread of human trypanosomiasis.

Swynnerton (1923) recommends attacking the game-tsetses by controlled and systematic grass-burning. This procedure is safe and promises good results. It allows of no circumvention by the fly, such as may follow attempts at wholesale destruction of the Big Game.

Judicious human settlement, with all that this entails of propaganda among the natives, fly-surveys, and partial clearing, should, when possible, be combined with the burning measures. For example, routine daily search for firewood by natives, in the vicinity of their villages, could easily be turned to valuable account. Most of the trees of the fly-scrub are of small girth; and if, while wandering about looking for dead wood, each individual could be persuaded to cut off at the ground level even two or three little trees, the combined effect would soon be very noticeable.

In *palpalis* areas the nature of the fly's habitat precludes our relying on grass-burning. The effect of clearing on this tsetse is, however, well known. Certain of the Sese Islands to-day afford a striking illustration. An extraordinary multiplication of the situtunga antelope, *Tragelaphus spekei,* has taken place on these Islands since the removal of the population in 1909. The browsing of innumerable antelope has resulted in a disappearance of undergrowth along the water's edge, to a height of several feet from the ground, and many stretches of shoreline once thickly fly-infested are now absolutely free. Mr Fiske's experiments with cattle and goats—at present in progress on selected islands—show that domestic animals are equally efficient in this respect.

By proceeding along such lines and utilizing natural resources wherever possible, we avoid the penalties so liable to follow attempts at drastic interference with Nature's arrangements. No doubt the game will ultimately

disappear before the advance of human settlement, but we are not justified in ruthlessly destroying an irreplaceable link with the past merely as a *placebo* to a clamorous public. Before taking this irrevocable step, let us give a fair trial to the relatively simple yet scientific methods proposed by Swynnerton.

CONCLUSIONS.

Three main conclusions emerge from this review.

(1) The first is that *the present system of classifying the mammalian trypanosomes is unsound.* It is also unnecessarily complicated. Many of the so-called species are in reality strains or races or varieties of a single wide-spread species, and the differences between them are determined by the different environments in which that species lives.

The virulence of a trypanosome is a physiological character which is readily influenced by the environment to which the parasite is exposed, and the same is true of the preferences and infectivity of a trypanosome for different mammalian hosts. *In the present system of classification an altogether exaggerated importance is allowed to physiological variations*, several of the so-called species being distinguished from one another solely by differences in virulence.

The characters upon which reliance can be placed in zoological classification are (1) *the morphology and* (2) *the developmental behaviour of the organism in the natural insect intermediary.* Agreement in these essentials should be regarded as proof of specific identity.

Applying these principles to the classification of the mammalian trypanosomes of Africa, we find that they fall into three main groups according to their developmental behaviour in the Glossina. We have referred to these as the "proboscis-only," the "proboscis-and-gut," and the "gut-and-gland" groups. Moreover, each of these groups possesses a characteristic morphology —the *vivax-uniforme* type, the *nanum-congolense* type, and the polymorphic type.

We are, accordingly, confronted with three true zoological groups of mammalian trypanosomes. In the "proboscis-only" group, there are indications that *T. vivax* and *T. uniforme* are specifically distinct. The two are different morphologically, and from experience with naturally infected wild flies I believe that the true anterior station of *T. uniforme* is the hypopharynx rather than the labrum. In flies carrying *T. vivax*, the labrum always contains clusters of large flagellates, very easily recognized; but in the course of dissections of wild flies another type of proboscis-infection is often met with in which a relatively small number of tiny trypanosomes are found only in the hypopharynx. These infections are, I think, ascribable to *T. uniforme*, and if this be confirmed *T. vivax* and *T. uniforme* may be regarded as "good" species.

In the "proboscis-and-gut" group we find "*T. nanum*" and "*T. congolense* (= *pecorum*)": and these, in my view, are physiologically different strains or varieties of a single species.

In the "gut-and-gland" group we have the polymorphic trypanosomes—"*T. brucei*," "*T. gambiense*," "*T. rhodesiense*." Here again, all the evidence points to the conclusion that these organisms are not severally distinct species, but physiological variants of a single species.

It is difficult to assign a place to the three pathogenic trypanosomes known as *T. evansi*, *T. equinum*, and *T. equiperdum*. These parasites do not rely on the Glossinae for their transmission, and their tendency towards monomorphism may be intimately associated with the loss of the power of cyclical development in an insect. A great deal of work remains to be done on these trypanosomes, and valuable light might be thrown on the ancestry of these forms if they could be persuaded to develop in Glossinae. For the meantime, I think they should remain as appendages of the polymorphic group.

(2) The second conclusion concerns the relation of the big game to the trypanosomes of man and of his domestic animals. On this subject we have said all that is at present necessary in section (9).

There is a tendency to assume that a trypanosome from man, when introduced into game, will retain its peculiarities if it is taken up again from the game by the fly. But the possibility that sojourn in the game exerts what may be described as a diluting effect on the trypanosome in reference to man, has not been sufficiently recognized, and it is hoped that the arguments advanced in this paper will draw attention to this aspect of the question.

(3) The third conclusion is that the possible importance of direct transmission in determining the virulence and spread of trypanosomes in nature has not, up to the present, received sufficient recognition.

There is good reason to believe that direct transmission plays a very important rôle in the spread of trypanosomiasis in human communities. The method by which the parasite is transmitted from man to man probably influences its virulence, and a general epidemiological survey of the areas where human trypanosomiasis exists suggests very strongly the conclusion that direct transmission is responsible for the disease in its epidemic form. The extent to which coincident diseases such as ankylostomiasis contribute to the virulence of the trypanosome in man is a problem that calls urgently for investigation.

I wish to say, in conclusion, that I owe a great debt of gratitude to Mr Clifford Dobell, F.R.S., for valuable suggestions as to the arrangement of this paper, and for his kindness in undertaking the onerous duty of preparing it for publication and seeing it through the press.

ENTEBBE, UGANDA.
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SPIRELLA REGAUDI IN THE CAT[1].

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(With Plates VIII—X including Figs. 1–23.)

The spiral organism found in the stomach of various carnivora has been investigated by several observers (see the references at the end of this paper). Such studies have been mainly directed to the morphology of the organism, its infectivity and the part it plays in producing a pathological condition. In the present paper I have been concerned rather with the physiological condition of its host as influencing the prevalence and location of the organism in different regions of the stomach. I was anxious further to ascertain if the organism affected the normal processes of digestion in the stomach either to the advantage or the disadvantage of the animal entertaining it.

I. THE REGIONAL DISTRIBUTION OF THE ORGANISM.

The organism is to be found both in the fundus end and the pyloric end of the cat's stomach. It appears to invade certain of the epithelial cells of the fundus glands but practically never passes through the basement membrane of the epithelium. It has a marked preference for the solid walls of the tubes or the surface-epithelium of the stomach and appears only to leave this shelter when somewhat thick mucus can give it harbourage. I have found no evidence that it will pass freely into the thinner liquids that may at times be present in the stomach. Fig. 1 (Pl. VIII) shows the organisms in the mouth of a fundus gland where two tubules open into a common mouth. Fig. 2 exhibits them in the mouth of a pyloric gland. In Fig. 3 they are shown in the neck region of a fundus gland and their connection with the so-called oxyntic cells is striking. This frequently described association with the oxyntic cells is seen in Fig. 4 (representing an oblique section of a gland) and Fig. 5. The organisms are practically never seen in the duodenum, even at the beginning. Though observable at the extreme cardiac end of the stomach they rarely invade the oesophagus. On only one occasion have I found them in the deeper side of the epithelium in the subepithelial lymphoid tissue and here they were located within typical cells of that tissue which presumably possess a phagocytic function. This is somewhat obscurely shown in Fig. 6. There is some difficulty in determining the precise nature of their association with the oxyntic cells

[1] The expenses of this investigation have been defrayed by a grant from the Medical Research Council.

at times. It must be remembered that histologists[1] frequently represent the normal mucous membrane as showing the oxyntic cells of the neck of the fundus gland as much excavated on the luminal aspect so that the lumen of the tube is pouched out laterally into the interior of many of the oxyntic cells. In section these pouchings may appear to be cavities in the oxyntic cells. Consequently organisms actually in the luminal bays may appear to be located in closed cavities in cells. Although this is frequently the case I am satisfied that at times the spiral organism is actually enclosed within the oxyntic cells.

II. DEGREE OF PREVALENCE OF THE ORGANISM IN CATS.

During the last two years I have examined over 105 cats particularly in connection with the conditions in which the organism appears. Some of these cats had fasted for 24 hours, some were in various stages of digestion. Different kinds of food were studied as regards effect on the presence of the organism; in some cats secretion was induced in the empty stomach by the intravenous injection of gastrin and in some cases operative procedures were adopted to afford material for studying the organism in the same animal at different digestive periods. The condition of the mucous membrane as regards healthiness was in all cases observed. The cases in which the organism was not found were almost invariably cats in the fasting condition. But I am in agreement with others who state that the organism is generally absent in very young animals. On the other hand, a cat in digestion almost invariably exhibits it in some form or other. The suggestion that has been advanced (Lim, 1920) that the presence of the organism is due to a local laboratory infection must be put aside. My animals were in a large proportion of cases in the laboratory only 24 hours and were derived from different sources. Some of the material I examined came from another laboratory and was prepared as long ago as 1912. It seems permissible to regard the presence of these organisms to be as characteristic of the cat's gastric mucous membrane as fleas are of its fur. I think, further, that it is justifiable to state that the presence of the spiral organism is, ordinarily, provocative of no obvious pathological change.

Of 105 cats which were examined, 99 were specially examined for the fundus end and in 78 cases the spiral organism was found. In 60 cats in which the pyloric end was specially examined the organisms were found in 32 cases. This includes cases of kittens of various ages which practically never show them. It is a fair estimate to say that the organism is present in at least 80 per cent. of cats in all stages of digestion and of all ages in the fundus end and in 50 per cent. of cats at the pyloric end. But, as will be seen later, the distribution in the stomach depends upon the digestive state. Of the cats examined by me a very large proportion were in the fasting condition.

[1] This is well shown in Bensley's figures in *American Journ. Anat.* II. (1902).

III. SOME POINTS IN THE ANATOMY AND PHYSIOLOGY OF THE ORGANISM.

The organism is a fixed but flexible spiral, the end turns have a decreasing diameter and the main filament terminates in a kind of apex situated in the axis of the spiral. There is attached at each end a flagellum. In nearly all cases that I have examined the spiral is right-handed, though in a small minority the spiral was possibly left-handed. The flagellum is also similarly a right-handed spiral; the pitch of this spiral is greater than that of the spiral filament, and the flagellum spiral forms approximately one complete turn. In the shorter organism formed from a recent transverse division of a longer form there is a flagellum at one end only (see Fig. 10, Pl. VIII). Nevertheless the organism can move quite freely forwards or backwards along its main axis. There is some kind of "jointing" at the attachment of the flagellum to the spiral filament since the flagellum may be turned right back so as to lie (with the same spiral turnings) against the main spiral (see Fig. 9).

The number of turns the main spiral filament possesses is variable. The organism may exhibit as many as sixteen or be restricted to two or three. Likewise the transverse diameter of the spiral and the thickness of the filament may vary. For reasons adduced later it seems that, whatever the size or thickness, we are dealing with merely one organism in different stages of development.

The fine filamentous form (found only in the fundus glands and never at the pyloric end of the stomach) measures about 3·4 to 4·0 μ for every six turns in length giving 0·6 μ per turn. The transverse diameter of the spiral is about 0·4 μ and the diameter of the filament is estimated at 0·08 μ. In optical section the turns are pitched at an angle slightly over 60°. Comparing these measurements with those of a maximally thick organism a length of five turns measures 6·0 μ giving 1·2 μ per turn; the transverse diameter of the spiral is not less than 0·85 μ and the diameter of the filament is estimated at 0·3 μ. Some may have thicker filaments even than this. The measurements were made from sections of formalin-Ringer fixed material stained with Giemsa. It must be emphasised that between these extremes, organisms may be found of all intermediate measurements in different regions of the stomach dependent mainly upon its precise condition as regards digestion[1].

(A dark-background microphotograph of a few of the larger form, found dead in some fairly clear mucus, is shown in Fig. 7.)

The thicker forms rarely have more than five or six turns.

[1] Mr Clifford Dobell has been kind enough to give me the measurements he has made by a method of greater precision than I employed. His material was fixed in Bouin's fluid and stained with iron-haematoxylin. He finds that the length per turn varies from 0·5 μ in the thinner forms to 1·0 μ in the thicker. The diameter of the spiral 0·5 μ in the thinner (giving a pitch of 60°) and 1·2 μ in the thicker. His estimate of the thickness of the filament is 0·2 μ in the thinner and 0·4 μ tapering to 0·25 μ in the thicker. The only important difference is in the thickness of the thinner spiral. My measurement was only a comparative estimate but I may perhaps have lighted upon some younger forms than he met with.

With respect to multiplication I have seen much evidence of transverse division, but never of longitudinal.

THE EFFECT OF ACIDS AND ALKALIES UPON THE ORGANISM.

The organism is extraordinarily resistant to considerable changes in the reaction of the medium. It seems clear that increase of hydrogen ion concentration up to a certain point intensifies normal movement. On the other hand, decrease does not easily abolish movement but the movements appear less "purposeful."

The method I have employed to examine the effect of reagents is as follows. A scraping of the mucous membrane is taken, gently spread upon a slide, and then, in succession, a few drops of the reagent are applied and poured off. Not much of the scraping is lost thereby. A final small quantity of the reagent is added, a cover glass applied and gently pressed down so as to spread out the scraping. The excess of liquid at the edges is dried off and melted paraffin applied over the edges by an ordinary pipe-cleaner. This avoids any disturbance of the specimen. It is now sealed in the medium to the exclusion of air.

In Ringer's fluid the organisms are seen, by dark-background illumination, in obvious movement, the shorter ones rather more vigorous than the longer, the movements of the latter being comparatively deliberate. In hydrochloric acid, $\frac{1}{100}$th normal (0·0365 per cent.) the rapidity of movement is somewhat increased. In decinormal hydrochloric acid (0·365 per cent.) the movement is optimal. All sizes of the organisms are found in rapid progressive movement, the flagella working in the most effective manner, and the vase-like figure formed by the posterior flagellum is at its clearest especially when an organism is seen passing over a dark part of the field. This movement continues for at least an hour. But it takes a long time for this strength of acid to bring about destruction of the organisms, assuming it ultimately does do so. I have put up such sealed preparations as above described and still found some movement shown 18 hours later. It seems fair to infer from all this that as the acidity of the stomach increases the organisms are influenced to move the more freely over the surface of the gastric mucous membrane. With caustic soda, $\frac{1}{100}$th normal, the movements are much the same as in Ringer's fluid. In 0·025 normal caustic soda (0·1 per cent.) movement is still clear for some time. But in both cases the movements are far from maximally vigorous. However, neither of these strengths of acid completely destroy the organisms as I have seen feeble flagellar movement in about 5 per cent. of organisms after 18 hours of exposure to these reagents. To summarise this it may be said that neither fairly strong acids nor alkalies readily abolish movement but the optimal conditions for progressive movement imply the existence of an acid reaction approximately that of the stomach in advanced digestion. The movement with fairly strong alkali is somewhat irregular and convulsive if at all vigorous, with strong acids of a strikingly effective order.

Movement of the Organism.

There is little doubt that the movements of all kinds which the organism may exhibit are the consequence of primary movement of the flagella. In progressive movement the posterior flagellum is an active agent. The main spiral body does not appear to take any active share in the movement. It is stated by Kasai and Kobayashi (1919) that every two consecutive turns are more closely spaced, and a shifting of this spacing along the organism, passing as a wave, could account for a progressive movement. But though some irregularity in the arrangement of the spirals may sometimes be seen, I am convinced that the rule is for perfectly regular spacing. One may occasionally observe boring movement of an organism through some material and then the forward end of the organism passes through exactly as a screw traverses a thin plank of wood. If one keeps one's eye on the thread nearest the mass being bored, it is difficult to appreciate any movement whatever, so regular are the turns. Further, this affords, by itself, conclusive evidence of the existence of revolution of the organism in such progressive movement. It seems therefore reasonably certain that the power for the movement depends upon the posterior flagellum, at any rate in part, and that the revolution of the body is the consequence of the resistance of the medium, *a fronte*, though this revolution may be further assisted by any revolving movement occurring in the flagella.

The flagella form, in what I regard as the typical condition, about one complete spiral turn, the diameter of this spiral being greater than that of the body. They are jointed to the apical end of the body in such a way that they may be bent round the body spiral, still maintaining their spiral dimensions. In the resting state they may both occupy an intermediate position with the axis of the flagellar spirals at right angles to the body spiral. This is a position generally shown in the quiescent or dead organism (see Fig. 8, Pl. VIII). But in the active condition (unless in convulsive movement from adhesion to some foreign substance, when the flagellar movements may become very irregular) one flagellum appears to be more or less rigidly extended at an apical end, the other is retracted around the body (see Fig. 9). As the consequence of some alteration in shape or position of probably both flagella the organism progresses, the extended flagellum being at the tail end. In rapid movements one can detect a certain amount of "flicker," and the revolution of the whole posterior flagellum is seen to trace out a vase-like figure.

In the slow movements that may be seen in expiring or fatigued organisms one possible cause of the normal movement is elucidated. One may see a slow circular revolution of the flagellum, still maintaining its form and rigidity, followed by a more rapid reverse revolution. This is best seen when the body of the main spiral is stuck so that no movement of rotation can occur in the organism as a whole. But if the base of the flagellar spiral is itself rotating as would occur if the whole organism were revolving such a change could only give an appearance of flicker to the flagellar revolution.

Perhaps one should not insist too strongly that the figure traced by the flagellar revolution should under these circumstances be clean cut, but the fact remains that such vase-like figure is somewhat blurred. This suggests that perhaps another mechanism is operating however.

One explanation of the blurring in this figure is that there is some slight change of form in the flagellar spiral. If, as in a cilium, the whole flagellar filament was made up of two components, a contractile element and an elastic element arranged alongside, and if, further, the contractile element was arranged so as to occupy the peripheral position in the spiral, then shortening of the contractile element would result in a change of shape of the spiral to a degree corresponding with the extent of contraction. In any case there would be some amount of uncoiling of the whole flagellar spiral dependent in quantity upon the degree of contraction. The uncoiling would tend to make the spiral approach a simple straight extension. The mechanical effect of this would be of the nature of a posterior push against the containing medium. Such slight uncoiling with a slower restoration, rapidly repeated as in a cilium, would be effective in promoting a forward movement, would also account for flicker and explain the blurred figure of revolution. It is difficult to satisfy oneself of the existence of this kind of movement in slowly moving flagella, but it is possible again that it may occur.

We have then two mechanisms possibly accounting for the "push" forward given to the organism by the posterior flagellum. If, after an interval, the posterior flagellum becomes retracted and the anterior extended, the conditions are reversed, and the flagellar movements will then cause the organism to move in the reverse direction.

When the anterior flagellum is retracted it lies against the body of the organism, preserving its original curves (Fig. 9, Pl. VIII); its filament now has its different arcs lying parallel to those of the extended posterior flagellum, and it remains a right-handed spiral. The two mechanisms above mentioned can still operate in the retracted flagellum as in the posterior extended flagellum and lead to movement in the same direction as is produced by that flagellum. Unless the flagella are simultaneously extended or retracted the movement of the one will assist that of the other. In other words, the retracted anterior flagellum acts as a "tractor," whilst the extended posterior acts as a "pusher." And if, after recent division, an organism has only one flagellum (Fig. 10) it can still move according to the position of the flagellum in one direction or the reverse as may be frequently observed.

One can assert with confidence that undulatory movement of the flagella, as occurs with the long flagella of many other organisms, are entirely absent in this organism. Moreover one cannot easily conceive how any uniform "circumductory" movement of the flagellum can be effective in promoting progression.

IV. THE EXTENT, SUPERFICIALLY, OF THE DISTRIBUTION OF THE ORGANISM.

At the latter end of digestion the organism may be found both at the fundus and the pyloric ends of the stomach. In such cases the question will arise whether, nevertheless, the distribution is patchy, or whether the organism is evenly spread over the whole surface. A careful investigation of the greater part of the whole surface of the stomach, in a case in which the organism was found at both ends and which represented a stomach taken at the latter end of prolonged digestion, showed that the organism was similarly distributed both along the lesser and the greater curvatures and was continuously present from the extreme cardiac to the extreme pyloric end. There was no evidence that portions of the mucous membrane could escape infection.

V. THE FUNDUS END IN DIFFERENT DIGESTIVE STATES IN RELATION TO THE PRESENCE AND CHARACTER OF THE ORGANISM.

I have met with no instance in which the organisms were to be found at the pyloric end but not at the fundus end. If they are observable in the stomach at all they will be found in the fundus mucous membrane. On the other hand, they may be rare in the fundus end and absent at the pyloric. (I here rely upon observation made after fasting periods not exceeding 24 hours.)

If the fundus mucous membrane be examined in the early stages of digestion the organism will generally be found on the surface of the mucous membrane in the mouths and necks of the fundus glands but not within the glands at a depth where the so-called peptic cells are first evident. In the neck region where the oxyntic cells are very numerous and line up to the boundaries of the lumen of the gland the organisms are at a maximum in prevalence. They are here constantly found lying in the bays existing in the oxyntic cells and give the impression of streaming out of the oxyntic cells. In early digestion they are of the fine filamentous variety, sometimes comparatively short with but few turns, and then it is probable that they are actually within the oxyntic cells, sometimes they may reach a length of 15 or more turns and then they seem to have evacuated the cells. They seem to be developing from small stainable rounded bodies located within the oxyntic cells. Fig. 11 shows a binuclear oxyntic cell in which organisms of some length and the small stainable masses are exhibited within a cell. Fig. 3 shows them passing from oxyntic cells into the lumen and becoming directed towards the mouth of the gland. Figs. 4 and 5 also illustrate these points. Passing to the mouths of the gland the organisms may be seen at different depths from the actual surface. Where they are in the deeper part of the mouth they are usually thin but as they approach the surface they gradually thicken and simultaneously tend to divide. This tendency to become thicker is shown in Fig. 12 though the rate of increase in thickness is usually not so rapid. At the actual surface of the fundus end there may usually be seen some organisms of intermediate thickness and of considerable length (10 or 12 turns), some shorter but thicker, but

none of minimal thickness. Transverse division is common, especially as the thickening becomes evident. There is considerable difficulty in determining, with any precision, the earliest stages of development. It is possible to make out, in sections of the mucous membrane in which no spiral organisms can be seen, small homogeneous spherical masses, staining hardly at all, in size less than 0·2μ, located in the oxyntic cells. But in other cases it is difficult even to see these. This may really mean that the particular animal is not infected, though in apparently non-infected animals it may be that the organism is in some concealed form or at any rate one that is not revealed by the ordinary microscopic methods. I am quite unable to put forward any view upon this point though it seems possible that the germ of the organism may be present in some form not revealed by the histological methods employed.

As regards the slightly staining masses referred to above, I am more inclined to regard them as the last stages of degeneration of broken-down organisms. Fig. 13 (Pl. IX) shows an oxyntic cell in which early stages of development are exhibited and this was photographed from the mucous membrane of a fasting animal in which gastrin had been intravenously injected and which duly reacted with a secretion of gastric juice.

The above remarks have reference to the usual appearance exhibited in the gastric mucous membrane during digestion or at any rate when digestion has not been abnormally protracted. But in the later stages of digestion another appearance is seen. The organisms present are much thicker and shorter. Both filamentous and thick forms may be simultaneously present, but as digestion becomes prolonged or tends to become completed finally only the thick form is seen. The thin forms in particular lose their homogeneity and deeply stained beads are shown by the ordinary basic dyes. These have been noticed by other observers and are referred to by them as "involution forms." This "beading" is mainly characteristic of the thin forms; the thick organisms have much less tendency to "bead." Fig. 14 shows a section of the fundus mucous membrane in which the organisms are all thin and in the beaded condition. The beading may be seen in organisms actually within an oxyntic cell but whether they have never left the cell or have re-invaded it is not clear. This is seen in Fig. 15. The further history of the beaded form seems to be that the spiral gradually straightens, becomes less definite, and the deeply staining beads lose their affinity for the basic stain and slowly become unrecognisable. With regard to the thick form it may sometimes be the case that this form alone is present. The mouth of the gland may be comparatively free, the neck containing many. They may be present even within the oxyntic cells (Fig. 16). An intermediate thickness may sometimes be shown in which beading is generally evident (Fig. 17). The thick spiral organisms eventually segment till small masses alone remain and these may be located either in the lumina as in Fig. 18 or within the oxyntic cells as in Fig. 19. At times small deeply staining masses may be seen between the basement membrane and the cell which appear to be relics of segmented organisms.

I have endeavoured to trace the changes I believed to occur during digestion by direct methods. I will postpone the description of the results of these until the appearance at the pyloric end has been described.

VI. THE PYLORIC END IN DIFERENT DIGESTIVE STATES IN RELATION TO THE PRESENCE AND CHARACTER OF THE ORGANISMS.

The organism is present in or on the pyloric mucous membrane in a majority of cases examined. As one approaches the duodenum its presence is less conspicuous, till at the final part of the pyloric region it is quite rare. In some cases it may be seen only in the surface mucous, sometimes in the mouth of the gland as well, at times even in the body of the gland. In all cases it is the thick form only that is seen, with comparatively few turns, varying from one or two to five or six. There is generally much evidence of transverse segmentation till the short fragments alone are conspicuous. The appearance in the opening of a mouth of a pyloric gland is seen in Fig. 20 (Pl. X). Either because the large funnel-shaped mouths of the pyloric glands trap them as they progress in the surface mucous or by reason of some chemiotactic attraction the interior of the glands exercise, they readily invade these pyloric glands, so that when gastro-duodenal junction is examined there are none left. At the position of the bifurcation of a mouth into two necks they are seen in Fig. 21, and actually in the body of the gland in Fig. 22. They ultimately break down into formless relics and finally resolve into granular débris. The organisms do not invade the epithelium as in the fundus end. This is to be explained by the fact that the attractive cells, viz. the oxyntics, are absent at the pyloric end. But cells which are to be regarded as the representatives of the oxyntics, the so-called "Nussbaum's cells," may occasionally contain them. In no condition of digestion can the fine filamentous form be found so that it must be held that the organisms never originate at the pyloric end. With the passing along of the food, aided by their own movements, they reach the pyloric end from the fundus end, pass into the pyloric glands and perish.

This account of the history of the organisms is supported by the appearances presented by the mucous membrane in the so-called intermediate zone. Here the way they seem to take refuge in the oxyntic cells is very striking and is shown in Fig. 23.

What should be taken as determining the limits of the different regions of the gastric mucous membrane is not agreed by all observers. Lim (1920) regards all that portion which exhibits oxyntic cells as fundus region, that part which does not show them as pyloric. He therefore restricts the pyloric region to a zone of one-half to three-quarters of an inch. I should prefer to lay stress upon the essential difference between the cells of the pyloric and the fundus regions, histologically and physiologically. In appropriately fixed tissue the chief cells of the body of the fundus gland show the so-called pepsinogen granules deeply, but somewhat metachromatically, stained with basic dyes. Oxyntic cells are also present. At about 8 cm. from the pylorus the bodies of the glands become more twisted, the mouth much wider. Here the oxyntic cells are still numerous but the peptic cells become gradually replaced by the clearer cells characteristic of the pyloric glands. This region of 2 or 3 cm.

is best described as the intermediate zone. Beyond this the peptic cells fail, oxyntic cells become rare and this should be designated the pyloric region (about 5 or 6 cm.). The histological appearances, on the whole, favour Nussbaum's cells being regarded as outlying oxyntic cells. In the above description I am referring to the stomach of the cat alone. For measurement, the muscular coat was first removed and the mucous membrane spread out upon a glass plate.

VII. THE RELATION OF THE PRESENCE OF THE ORGANISM TO THE DIGESTIVE STATE AS DETERMINED BY EXPERIMENTAL METHODS.

In one set of experiments I simply fed a fasting animal with prescribed food and killed it by concussion and pithing after a certain interval. In another set the animal was anaesthetised, the oesophagus ligatured, a tube was tied into the stomach at the cut pylorus and different foods introduced through this tube. In other experiments gastrin was injected into the femoral vein, the stomach remaining empty. In still other experiments, after a preliminary anaesthesia during the operative procedure, the animal was decerebrated by Langley's method. In a further set a method was adopted which allowed samples of the mucous membrane to be taken during digestion.

In these experiments of serial observation, after the preliminary operation, a piece of the wall was raised by toothed forceps, pinched off with curved Spencer Wells forceps and cut away closely along the edge of the S. W. forceps. The S. W. forceps were arranged to interfere with main vessels as little as possible. In this manner a small piece of the whole thickness of the wall was obtained, measuring about 2 by $\frac{1}{2}$ cm. The cavity of the stomach remained closed since the S. W. forceps were not removed. As many as four such samples have been taken during an experiment. The piece removed was at once placed in the fixing fluid, except for a fragment of one end which was teased and examined by the dark-background method.

In all these experiments the results were essentially similar. The actual stimulus of food was not absolutely necessary for the development of the organism; the secretory state seems the essential thing as demonstrated in the gastrin cases. Flesh food seems more successful than milk for production, and the acid in these flesh-feeding cases reaches a higher level than with milk. In many cases the hydrogen ion concentration of the gastric contents was estimated by indicators. A much higher acidity was reached after gastrin in decerebrate animals than with animals kept under urethane anaesthesia, and some parallelism was frequently observed between the degree of acid and the quantity of organisms present though not invariably.

GENERAL CONCLUSIONS.

The precise appearance that will be presented by the gastric mucous membrane of any given cat cannot be predicted with certainty. It will vary according to the period that has elapsed since the last meal, it will be influenced by the intensity of the animal's gastric activity, and it will depend probably upon the degree of the animal's infection. In the fasting animal it will almost invariably be found that the number of organisms present is far from maximal

and that the fine filamentous form is comparatively rare. In the fasting state it may be a matter of difficulty to find more than two or three in a section. At times none at all may be discovered, though this may be due to the animal having escaped infection. In all my cases in which the organism was present in great abundance and in all varieties the secretory activity of the stomach was well established. I emphasise also that it is not the mere presence of food but the existence of the secretory process that is apparently the determining factor. Stress must also be laid upon the fact that only the thicker form is to be found at the pyloric end; at the end of active digestion the thicker form may be present at the fundus end and thick organisms may be seen in large numbers even within the fundus glands. After the examination of a large number of animals I conclude the typical condition to be as follows:

(i) *Early digestion.* Fine filamentous forms in the oxyntic cells (mainly of the neck) and in the lumina of the glands, becoming thicker as they pass up the mouths and reach the surface. No organisms at the pyloric end.

(ii) *Late digestion.* Filamentous forms rarer at the fundus end, thick forms at the pyloric end.

(iii) *Post digestion.* Filamentous forms absent, diminishing numbers of thick forms at both fundus and pyloric ends, a marked tendency for these to break down. Where oxyntic cells are present these post-digestion forms appear to seek harbourage in such cells, but nevertheless break down.

Flesh food seems to be more effective than other less natural foods in promoting development of the organisms.

I have made a certain number of attempts to breed these organisms *in vitro*, but with no more success than other observers. Struck as I was with the extraordinary effect of strong acids upon the organism I aimed at getting evidence upon the possibility of it contributing to the production of the acid which is so powerful in the gastric juice of the carnivora. The utmost I could find was some parallelism between the degree of acid and the abundance of organisms but quite inconclusive as evidence of the production of acid.

In conclusion it is my pleasing duty to record my gratitude to Mr Clifford Dobell in the more morphological part of this work. I cannot easily express the value to be attached to the criticism and guidance which he has been kind enough always to place at my service.

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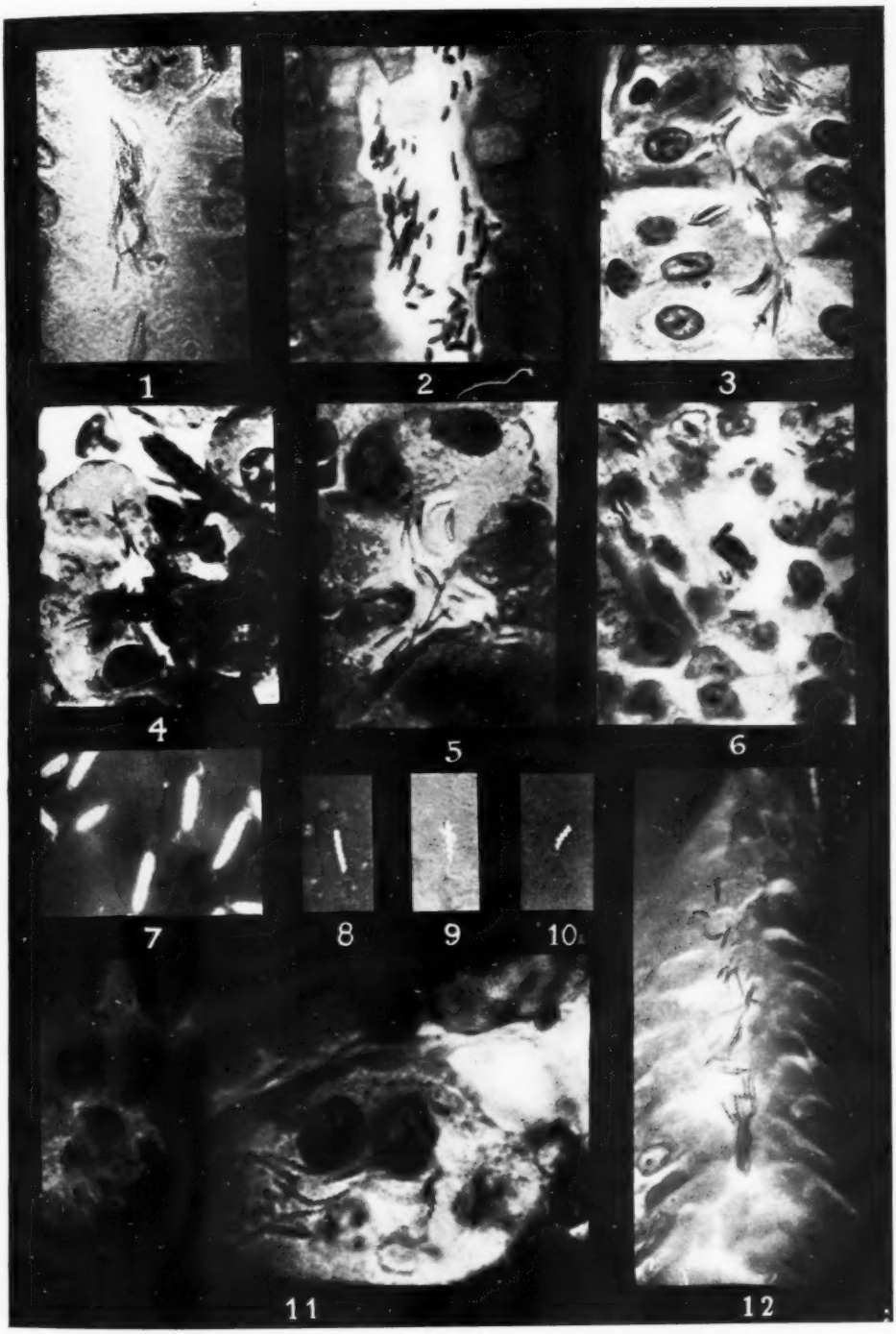
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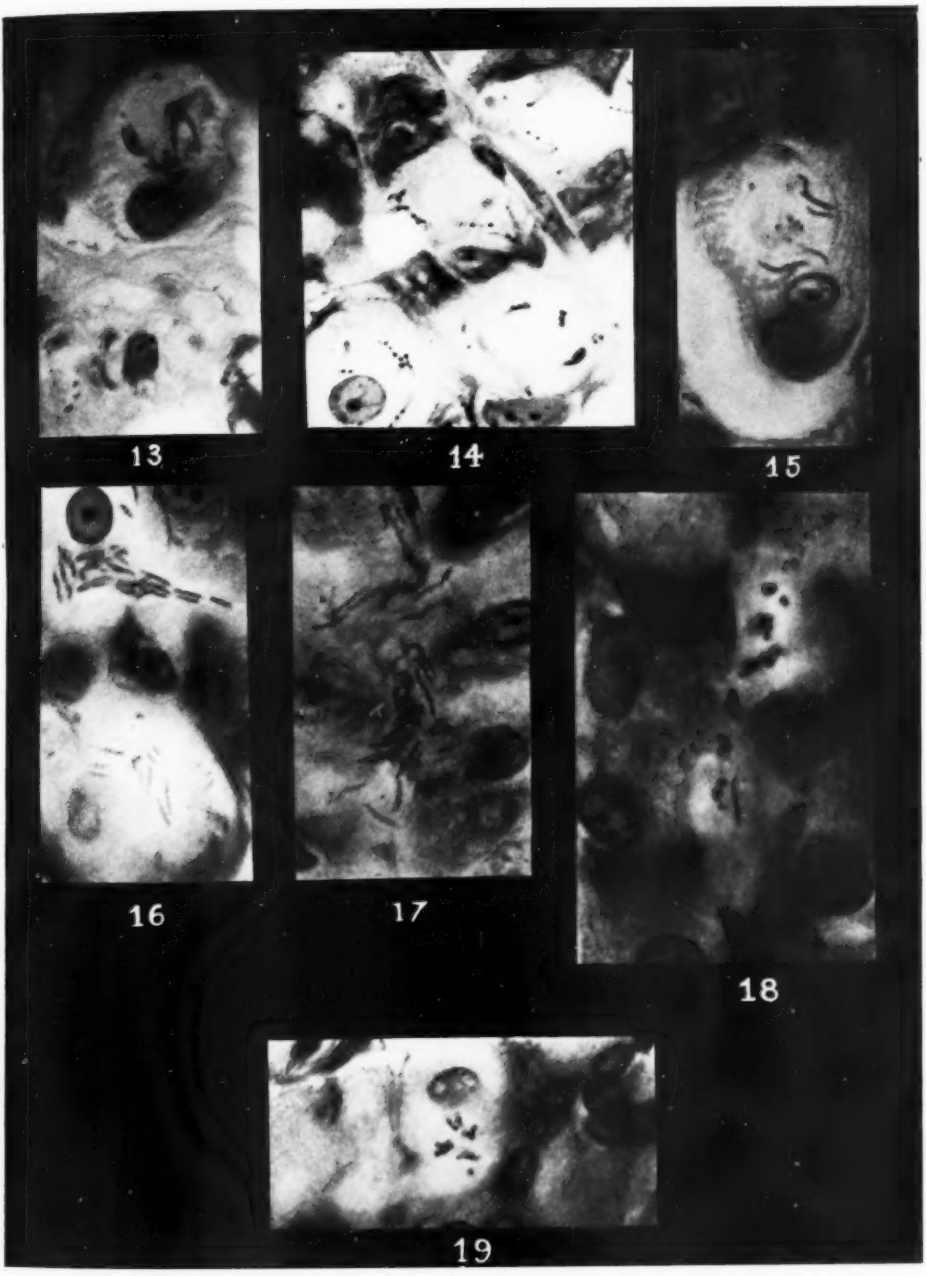
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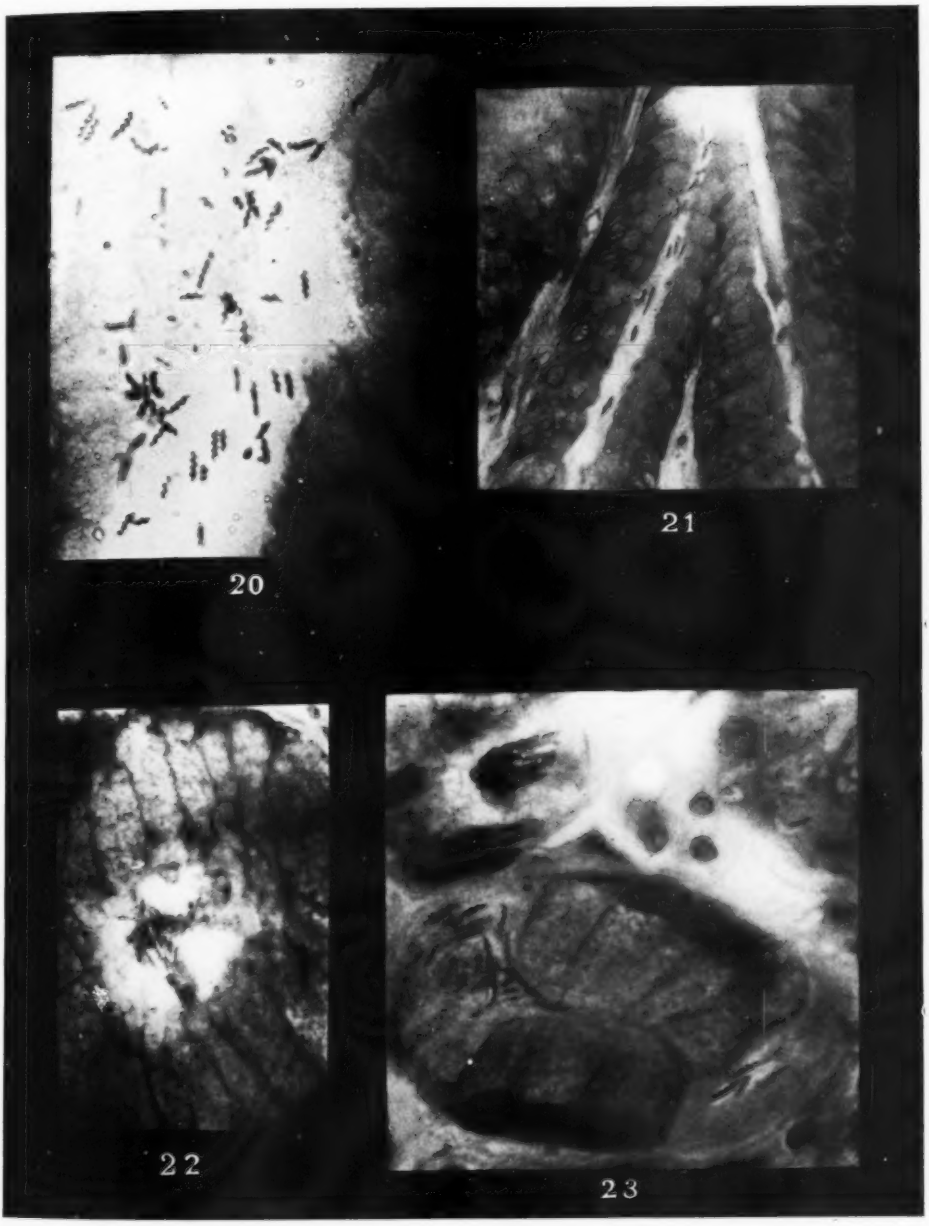

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DESCRIPTION OF PLATES VIII—X.

All the microphotographs were made with either a Leitz $\frac{1}{12}$, N.A. 1·3, Ocular iii, or a Zeiss Apochromatic 2 mm., N.A. 1·3, Oil Immersion, Compens. Ocular No. 6. The length of the camera shaft was such as to give a magnification of about 800 in the lower and exactly 1300 in the higher magnification. As source of light a Lilliput arc lamp with a Wratten filter *E* or filter *K*3 was employed.

PLATE VIII.

Fig. 1. Two fundus glands opening into a common mouth. Toluidine blue.
Fig. 2. Mouth of a pyloric gland. Giemsa.
Fig. 3. Neck of a fundus gland. Silver impregnation and Giemsa.
Fig. 4. Oblique section of fundus glands. Giemsa.
Fig. 5. Transverse section of fundus glands. Giemsa.
Fig. 6. Sub-epithelial tissue at fundus end. Giemsa.
Fig. 7. Dark-background photograph of fresh mucus.
Fig. 8. Film preparation stained Benians' relief method.
Fig. 9. Film preparation, relief staining, showing extended and retracted flagella.
Fig. 10. Film preparation, relief staining, recently divided organism with one flagellum.
Fig. 11. A binuclear oxyntic cell showing early stages of organism. From a gastrin experiment. Giemsa.
Fig. 12. Mouth of a fundus gland showing increase of size of organisms on approaching surface.

PLATE IX.

Fig. 13. Early stages of organism. Giemsa.
Fig. 14. "Involution forms" of organism within oxyntic cells. Toluidine blue.
Fig. 15. "Involution forms" in oxyntic cell. Giemsa.
Fig. 16. Thicker forms of organism within oxyntic cells. Giemsa.
Fig. 17. Organisms of intermediate thickness in the lumen of a fundus gland. Giemsa.
Fig. 18. Segmented relics of organisms within the lumen of a fundus gland. Giemsa.
Fig. 19. Segmented relics within an oxyntic cell. Giemsa.

PLATE X.

Fig. 20. Thick organisms in mouth of pyloric cell. Giemsa.
Fig. 21. Organisms streaming up pyloric glands from a common mouth. Giemsa.
Fig. 22. Thick organisms within the body of a pyloric gland. Giemsa.
Fig. 23. Transverse section of a gland from the intermediate region of stomach, showing thick organisms harbouring within oxyntic cells. Giemsa.

A PROTOZOOLOGICAL BICENTENARY: ANTONY VAN LEEUWENHOEK (1632–1723) AND LOUIS JOBLOT (1645–1723).

By CLIFFORD DOBELL, F.R.S.

The present year, 1923, has already witnessed the celebration of more than one scientific centenary. From divers parts of the world men have gathered together to do homage to Louis Pasteur, who was born just over 100 years ago; to Edward Jenner, who died just 100 years ago; and to Christopher Wren, who died just 200 years ago. But probably only a few of those who joined in these public thanksgivings recalled that 1923 is also notable, in the history of science, for another reason. Exactly two centuries have now passed since the death of the first and greatest of all "microbiologists"—Antony van Leeuwenhoek, Father of Protozoology and Bacteriology, the man who discovered those "microbes" now called protozoa, bacteria, and yeasts, which the illustrious Pasteur studied to such good purpose, and with which he laid, in part, the foundations of his scientific reputation: and it is exactly 200 years also since the death of another precursor of Pasteur—his countryman Louis Joblot, whose name is now almost forgotten, though it deserves to be remembered by all protozoologists.

Two hundred years is a long time in the history of modern science. Much can be learned in such a period, and much can be forgotten: perspectives can become inverted, and magnitudes can alter. Had we been alive in 1723, and could we have looked into the future, we should doubtless have regarded 1923 as a suitable occasion for paying tribute to the memory of Leeuwenhoek: and the more perspicacious among us might even have observed that the moment would also be opportune to mention the name of one of the more successful of his followers—Pasteur. To-day, however, we glorify Pasteur and recall dimly—if at all—that Leeuwenhoek was one of his "precursors." But I shall not discuss whether Pasteur followed Leeuwenhoek or Leeuwenhoek preceded Pasteur. It is so much a matter of standpoint in the observer. You look through one end of the telescope of time and behold an object large and near: but turn the telescope about, and look again, and the same object becomes afar-off and shrunken to insignificance. Meanwhile the object has suffered no change, and far be it from me to pronounce which of the two visions reveals it in its truer proportions.

My only aim in writing the following inadequate notes is to jog the memories of those who to-day use microscopes and study "microbes"—to

remind them, for a moment, of the lives and labours of two of their predecessors in the same field of work. Let us not forget them. Let us honour their memories and gratefully acknowledge our debt to them: and while others are publicly engaged in raising monuments to Pasteur, Jenner, and Wren, let us here, with all humility, in the comparative privacy of *Parasitology*, affix a little tablet to the memory of Leeuwenhoek and Joblot.

ANTONY VAN LEEUWENHOEK (1632–1723).

Leeuwenhoek has already received sympathetic notice in these pages from Professor Nuttall[1]. His portrait[2] has also been reproduced here, and will be familiar to almost every reader. The following notes are therefore merely supplementary to those which have previously appeared.

It is almost impossible, for anybody who has any knowledge of his works, to write about this remarkable man without enthusiastic admiration. Only one writer—an envious contemporary fellow-countryman—has tried to do so: and he merely succeeded in blackening his own character. This old Hollander was, in truth, one of the most original men who ever lived. It is impossible to compare him with anybody else, for he belongs to a genus of which he is the type and only species. Your typical scientific genius gets a good education: Leeuwenhoek got practically none. He goes to a university and studies under dustinguished professors: not so Leeuwenhoek. He imbibes the traditional knowledge of centuries and then begins to re-arrange and develop it for himself: but all that Leeuwenhoek knows he has learnt himself from Nature, and when he sets to work he relies entirely upon his own native genius. He cannot even read what others have written on the subjects that interest him. He can only talk to Nature, ask her questions in 17th-century Dutch, and puzzle out her answers by himself. How he did it is one of the "*onsigtbare verborgentheden*" which even he himself could not unveil to us.

Leeuwenhoek's life is soon told. He was born at Delft, in Holland, on 24 October 1632, in "the glorious age of Frederick Henry" which gave Holland and the world so many great men. He died there on 26 August 1723[3], aged nearly 91 years. He came of good Dutch stock, and was outwardly a simple good Dutch burgher. In his youth he had a commercial upbringing as bookkeeper in a draper's shop in Amsterdam; but at the age of barely 22 he married and settled in his native town, where he remained for the rest of his life. During the greater part of this time he held the post of Chamberlain to the Sheriffs of Delft—an office which was probably a sinecure—and devoted himself to microscopic studies. He was a well-born and probably well-to-do

[1] *Parasitology* (1921), XIII. 398–400.

[2] Prof. Nuttall—misled by the erroneous statements of others—notes that this portrait was executed in 1695, and shows Leeuwenhoek at the age of 63. It was really made about 1685, however, and consequently depicts him when he was ten years younger.

[3] Not 1725, as stated—by an unfortunate misprint—on the portrait which appeared in Vol. XIII. of *Parasitology*. (See correction, *ibid.* p. 398.) G. H. F. N.

provincial gentleman of Holland, and not a village beadle of "Jewish-Saxon" extraction as Richardson and Plimmer—on worthless evidence—would have us believe. His private life need not detain us here, however: at the moment we need only recall some of the things that he did.

For 40 years Leeuwenhoek remained in obscurity. During this time, however, he was not idle: he was learning, among other things, how to grind and mount lenses. In 1673 his fellow-townsman, Dr Reinier de Graaf—whose brief but brilliant career still has its memorial in the "Graafian follicle"—introduced him to the Royal Society of London: and thereafter, till the day of his death, Leeuwenhoek became one of the Society's most copious and honoured correspondents. He was elected a Fellow[1] of the Society in 1680, when he was 47 years of age, and this unexpected honour appears to have acted on him as a powerful incentive to continue his researches.

Leeuwenhoek did not get a good education, even for those days, for he never learnt Latin or Greek or any language but his own—and that not overwell, though it is a mistake to call him "illiterate." He was self-educated, and he worked and thought alone. Most of what he knew of the learning current in his time, he picked up in conversation with his friends. So far as his own work was concerned he was entirely independent. He began at the beginning—devised and made his own apparatus, carried out his experiments and observations in his own way, and drew his own conclusions from them. He possessed extraordinary perseverance and much ingenuity, and usually repeated and varied his experiments again and again in order to convince himself of their accuracy; and it was not until he was a very old and rather disappointed man that he ceased to take delight in demonstrating his discoveries to others. When he recorded any of his researches, it was invariably in the form of a letter—written to the Royal Society or some distinguished personage. These letters—of which about 200, written during the last 50 years of his life, are still in existence[2]—contain accounts of an enormous number of discoveries, mostly made with the aid of the microscope. They are written in a homely and unaffected style, in the common colloquial speech of his age and country. Generally they are long and rambling, and his thoughts come tumbling out with disconcerting inconsequence. What Leeuwenhoek knew about any subject is usually to be found in many different letters, often written at widely different dates, and is imbedded among masses of information and speculations on all manner of other subjects. These letters are not scientific memoirs but familiar conversations, and they convey an indescribable impression of the personality of the speaker. It is impossible to read them without

[1] Leeuwenhoek was a Fellow—not a Foreign Member—of the Royal Society (cf. *Parasitology*, XIII. 399). It is noteworthy that Christopher Wren was born in the same year as Leeuwenhoek (1632) and died in the same year (1723); and Wren was elected President of the Royal Society in the same year (1680) that Leeuwenhoek was elected a Fellow. They never met, apparently, though letters passed between them.

[2] The number of the extant letters of Leeuwenhoek is usually grossly overstated—some writers magnifying them to even twice their real number.

feelings of friendship for the man himself. His eye is so sharp, his head is so clear, his honesty and his sincerity are so transparent, that you unconsciously forgive his shortcomings and find excuses for his mistakes. He complains that he has "no style or pen," and no language but "the mother-tongue to which he was brought up": yet with such poor tools he succeeds, unwittingly, in hewing an image of himself such as few masters of the craft of letters could equal.

Leeuwenhoek's originality and uncommon ability are well exemplified in his microscopes, nearly all of which are, alas! lost, though several accounts of them survive. They were not microscopes—in the ordinary sense—at all, but simple magnifying glasses, consisting usually of a single minute but excellently wrought biconvex lens mounted between two flat metal plates. The object was adjusted before the lens by an awkward arrangement of screws. Every part was made by himself—some of his lenses, indeed, having been mounted in silver which he himself extracted from the ore. Nobody else has ever made "microscopes" like this, before or since; and it is doubtful whether any modern worker could do much with such instruments, though in Leeuwenhoek's own hands they were astonishingly efficient. He made and mounted more than 400 lenses in this fashion, and we know that some of them had a magnification of about 300 diameters, and gave excellent resolution; but how he succeeded in seeing some of the things which he describes—and with sufficient accuracy to vouch for his veracity—we shall probably never know. He never divulged the method which he used for making his "best observations," saying that he "kept that for himself alone."

There is no space to describe here all Leeuwenhoek's multifarious microscopic activities. I should like to tell of his demonstration of the capillary circulation, and of his comparative studies of the blood-corpuscles in many different animals: of his studies of the histology of all manner of animals and plants, and more particularly of his pioneer observations on the spermatozoa—so admirable, but still so persistently misunderstood: of his observations on the life-histories and the anatomy of insects—especially the louse, the flea, and the ant, and of his discovery of parthenogenesis in the Aphids: of his discovery of *Hydra*, and its reproduction by budding: of his discovery of the viviparous reproduction of the vinegar-eel: of his discovery of the Rotifers, and their remarkable power of surviving desiccation: of his contributions to animal and vegetable embryology, to crystallography and the microscopic study of chemical compounds: but it is impossible to discuss these and his many other discoveries here—even if I were competent to do so—and I must confine my attention to Leeuwenhoek's work on protozoology and bacteriology. Here, too, even the bare list of all his achievements would be amazing.

It was probably in 1674 that Leeuwenhoek first saw living protozoa. In the following year he observed and studied many species in rain-water, well-water, and canal-water, in infusions of pepper, ginger, and nutmegs, and elsewhere; and many of the organisms which he so quaintly described (*Vorti-*

cella, *Stylonychia*, *Bodo*, etc.) are still easily recognizable by anybody familiar with the free-living Protozoa. In the same year (1675) he discovered the Bacteria in waters of various kinds, and shortly afterwards recognizably described them. In 1674 he saw—though he did not know what they were—the first parasitic protozoa (oocysts of *Eimeria stiedae*): in 1681 he discovered the first entozoic protozoa (*Giardia*) ever found in man: and in 1683 he discovered and described the ciliates of frogs (*Opalina*, etc.). In 1680 he discovered anaerobic bacteria[1] in pepper infusions, and in the letter describing them recorded the first microscopic observations ever made on the yeasts in beer: in 1681 he discovered the bacteria of the human mouth and intestine—including the spirochaetes. In later years he not only discovered and described many more free-living protozoa, but he also studied their structure and development. He discovered *Haematococcus* (*Sphaerella*)—and other related flagellates—and ascertained its method of multiplication. He investigated *Vorticella* more thoroughly, and discovered and studied *Carchesium* and *Anthophysa* and *Kerona* and *Volvox*; and in the case of the last, he even discovered its remarkable mode of reproduction. He saw the cilia and cirrhi of ciliates, and the flagella of flagellates, and he even observed the conjugation of the Ciliata and interpreted it as "copulation."

Leeuwenhoek studied the Protozoa and the Bacteria with evident delight, and called them all "little animals," "beasties," or "little creatures" (*dierkens*, *beesjes*, *cleijne schepsels*). It appeared to him obvious that they must be organized in the same way as larger animals, and this often led him to make curious comparisons and sometimes to draw ludicrous deductions—as, for example, when he gravely calculated, with much arithmetic accuracy, the probable magnitude of the capillary blood-vessels in a bacterium. But he never professed to have seen such things, and he always drew a clear distinction between his observations and his inferences—his accounts of the former being almost invariably prefixed with "I have observed...," and of the latter with "but I imagine" or "I figure to myself." As a specimen of Leeuwenhoek's protozoology let me quote his account—the first ever given—of *Vorticella*, which he discovered in standing rain-water in 1675. He calls it "the little animal with a tail," and describes it thus[2]:

> Of the first sort of creatures that I discovered in this water, I saw, after divers observations, that the bodies consisted of 5, 6, 7, or 8 very clear globules, but without being able to discern any membrane or skin that held these globules together, or in which they were inclosed. When these little animals moved themselves, they sometimes stuck out two little horns[3], which were continually moved, after the fashion of a horse's ears. The part between these little horns was flat, their body else being roundish, save only that it ran somewhat to a point at the hinder end; at which pointed end it had a tail, near four times as long as

[1] As Prof. Beijerinck (1913) has recently shown in an admirable essay.

[2] From Letter dated 9 Oct. 1676. A partial translation was published in *Phil. Trans. Roy. Soc.* 1677.

[3] The optical section of the wreath of cilia round the peristome—so interpreted by most of the early observers.

the whole body, and looking as thick, when viewed through my microscope, as a spider's web[1]. At the end of this tail there was a pellet, of the bigness of one of the globules of the body; and this tail I could not perceive to be used by them for their movements in very clear water. These little animals were the most wretched creatures that I have ever seen; for when, with the pellet, they did but encounter any particles or little filaments (of which there are many in water, especially if it hath but stood some days), they stuck entangled in them; and then stretched their body into an oval, and did struggle, by strongly extending themselves, to get their tail loose; whereby their whole body then sprang back towards the pellet of the tail, and their tails then coiled up serpent-wise, after the fashion of a copper or iron wire that, having been wound close about a round stick, and then taken off, retained all its windings. This motion of extension and contraction of the tail continued; and I have seen several hundred of these little animals, caught fast by one another in a few filaments, lying within the compass of a coarse grain of sand.

Leeuwenhoek made many more observations on Vorticellids later, and finally reached much more satisfactory conclusions regarding their "tails" and "horns": but it would take us too far afield to discuss all this here. I must "break off"—as he so often says at the end of his letters.

In the foregoing notes I have mentioned only a few of Leeuwenhoek's discoveries in microbiology. Their outstanding merit is that they are original. Nobody before Leeuwenhoek had ever seen protozoa or bacteria[2]; yet he not only discovered them—by his own unaided efforts—but he also investigated them with all the means at his disposal. He was the first bacteriologist and the first protozoologist, and he created bacteriology and protozoology out of nothing. He did not realize the full importance of his discoveries—nor did anybody else in his day. But he was content to discover and observe, and verify his discoveries and observations by experiment; so that when, in his old age, he met with contradiction and even censure and ridicule, he said simply "It doesn't bother me: I know I'm in the right[3]."

It is hardly possible to do justice to Leeuwenhoek in a few lines. To appreciate him you must read what he himself wrote, and you must also know what he was writing about. The Latin translations of his letters are often faithful in substance, but they have not the flavour of their Dutch originals. The English versions of his writings published in the *Philosophical Transactions*[4] are also often faithful, but usually mutilated; while Hoole's *Select Works*, though usually good, are often too good—being re-arranged and carefully castrated for family reading. Yet it is from these versions that Leeuwenhoek is generally known outside of Holland. His manuscripts, which are not always

[1] *i.e.* looking as thick through the microscope as a spider's web appears to the naked eye.

[2] I am well aware, of course, that claims have been made for earlier workers; but all of those which I have examined appear to me to be wholly without foundation.

[3] "*Maar ik en stoor mij sulks niet, ik weet dat ik de waarheijt hebbe.*" (Letter of 26 Aug. 1717, to Boerhaave.)

[4] Not in the *Proceedings* of the Royal Society—as stated previously in *Parasitology* (1921), XIII. 398. The *Proceedings* did not make their appearance until many years later. It may be added that a number of Letters published incompletely in English in the *Phil. Trans.* have never yet been printed elsewhere, and are not to be found in Leeuwenhoek's collected works (Dutch or Latin).

easy to decipher, are not accessible to everybody; but they contain much that has never been published, and are often more informing than his printed works. It has been my good fortune to be able to study them, and consequently to learn to know Leeuwenhoek rather more intimately than most people; and if any reader of these lines should think that I exaggerate his merits, I would therefore beg him to study his Leeuwenhoek anew—in the original Dutch: to think upon the state of science in his day: and to remember who he was. I am confident that any such reader will then understand and share my admiration for this remarkable man. As I said at the beginning, it is almost impossible, for anybody who knows him, to write about Leeuwenhoek without enthusiasm.

There is not room, in this brief notice, to cite chapter and verse for every statement that I have made; but I would warn all who desire to know more of Leeuwenhoek and his work that, in so far as his life is concerned, his Dutch biographers alone are to be trusted. The fullest and most authoritative biography is that of Haaxman (1871, 1875); but much that has been written about Leeuwenhoek by English, French, and German writers is lamentably inaccurate, as also is the information contained in most biographical dictionaries. Haaxman quotes all his sources, and additional references of value will be found in Harting (1850, 1876). I cannot recommend the reader to study any other works—except those of Leeuwenhoek himself.

LOUIS JOBLOT (1645–1723).

All that is definitely known about Joblot[1] can be told in a very few words. He was born at Bar-le-Duc (Meuse) in 1645, though the exact date of his birth is unknown; but as he was baptized on August 9 in that year, it was probably early in August. His father was named Nicolas Joblot, and his mother's maiden name was Anne Guilly; and little "Louys"—as he was christened—was the fourth of six children resulting from their marriage. The Joblots appear to have been well-to-do citizens of Bar, but Nicolas's status is not known.

Of Louis Joblot's early years and upbringing we know nothing, though he evidently received a good education somewhere. The first 35 years of his life are a complete blank: but on 24 February 1680 he was appointed to the post of Assistant Professor of Mathematics, Geometry, and Perspective at the Royal Academy of Painting and Sculpture in Paris; and on 4 July 1699 he became Professor of the same subjects in succession to Sébastien Le Clerc. He occupied this chair until 1721, when he retired with the rank of Emeritus Professor. During his tenure of office he lectured on perspective, geometry, vision, the theory of light and colours, and the anatomy of the eye; and during this time also he devoted his attention to the study of magnetism, optics, and microscopy. With the assistance of a scientific-instrument maker in Paris, he

[1] Practically everything that we know concerning Joblot himself has been collected by Konarski (1895) and Brocard (1905), to whose works the reader may be referred for further details.

invented and constructed microscopes of various patterns, and then employed them in the investigation of microscopic animals. On 27 April 1723, when he was in his 78th year, Joblot died at Paris—apparently rather suddenly, for there is a record of his attendance at a meeting on April 3.

Apart from the information that can be gathered from his writings, this is practically all that is now certainly known about Joblot. He corresponded with several more or less well-known men of learning, but only a single manuscript of his—a letter to François Lamy now in the Bibliothèque Nationale (reproduced in facsimile by Brocard)—is known to exist. Moreover, no portrait of him is known, unless the rather crude picture of a microscopist at work, which heads the sections of his only book, can be regarded as a portrayal of himself. Probably he never married, and left no direct descendants; but this is merely an inference from negative evidence.

Joblot's title to scientific fame rests upon the evidence contained in his book on microscopes, published in 1718. It is a most curious production, and merits more attention than it has hitherto received. The book is divided into two parts—the first (78 pages and 22 plates) giving an account of his microscopes, the second (96 pages and 12 plates) being a record of his "new observations" made with these instruments "on an innumerable multitude of insects, and other animals of divers species," which occur in water and various infusions. Many of these "insects," or "fishes" as they are also termed, were protozoa: and this second part of Joblot's volume is, in fact, the first separate treatise ever written on the "Infusoria." It contains the first account, and the earliest published figures, of many different protozoa—some of them easily recognizable, but a number not identifiable with any certainty. The figures are mostly crude, and sometimes highly imaginative.

This singular work was published at the author's expense and the edition was probably small, for it is now extremely rare[1]. Unfortunately for Joblot, a second edition was published long after his death (1754), and this was edited and added to—from Joblot's MS. remains—by the publisher. This second edition is not uncommon, and has been consulted by most of those who have written on the history of protozoology. It contains nothing on the Protozoa that was not published in the first edition[2], but the difference in date detracts considerably from the apparent value of the observations. What was original and remarkable in 1718 appears somewhat commonplace for 1754. The following notes all have reference to the *editio princeps* of 1718.

Most of Joblot's observations were made—as already noted—upon organisms which he found in various vegetable infusions. Some of them had been described and figured before by others; but Joblot makes no mention of the fact, and records most of his forms as "new." He knew no names for them,

[1] I do not know of the existence of more than about half a dozen copies, though doubtless others are extant; but there is one in the British Museum, and another in my possession.

[2] The additions consist largely of material taken bodily out of other early works on micrography—often incorporated without acknowledgement.

and so was driven to invent more or less suitable designations himself. Thus we find various ciliates called "bagpipes" (*cornemuses*), "funnels" (*antonnoirs*), "kidneys" (*rognons*), "gluttons" (*goulus*), "pirouette dancers" (*sauteurs à la pirouette*), and so forth. Yet some of Joblot's fanciful "unscientific" names have survived to the present day, among them being "the slipper" (*chausson*) for *Paramecium*, and "the swan" (*sygne*) for *Amphileptus*. He often has several names for the same form, and sometimes gives the same name to different organisms, while his words and figures are sometimes extravagant: consequently it is by no means always easy to interpret his findings.

Apart from the new, or nearly new, organisms described by Joblot, the chief interest of his work lies in the experiments which he performed on the subject of "spontaneous generation"—a doctrine which he declares to be inconceivable, and contrary to all reason and religion. His own experiments—considering when they were made—are really remarkable. He found that an infusion of hay, made with cold water, was swarming with "as many as five or six sorts of living animals" (also called "insects" or "fishes") within a few days[1]. He then made another infusion, and divided it into two separate lots: one part was left exposed to the air, the other was put in a tightly stoppered vessel. Subsequent examination showed him that animalcules were produced equally in both; and he therefore made a new experiment which is so remarkable that I cannot refrain from quoting it bodily: "On the 13th October [1711]," he writes, "I boiled some similar fresh hay in ordinary water for more than a quarter of an hour. Afterwards I put equal quantities of it into two vessels, of approximately the same size: one of them I stoppered immediately, and even before it was wholly cooled; the other I left uncovered. In this I found animals at the end of several days, but not one in the infusion that had been stoppered. I kept it thus closed for a considerable time, in order that I might discover any living insects, if they should have appeared in it; but having found none of them, I finally unstoppered it, and at the end of several days I then found some in it. And this shows us that these animals had developed from eggs dispersed in the air, since such as might have happened to be on the hay had been completely destroyed in the boiling water[2]." Finally, after remarking that in his opinion he has made "a sufficient number of experiments to show that neither alteration, nor corruption, nor a bad odour, is the cause of the generation of these animals," Joblot advances the following "hypothesis" in explanation of the phenomena observed: "I shall suppose," he says, "that in the air, in the neighbourhood of the earth, there fly or float a countless number of very little animals of divers species, which settle upon those plants which are agreeable to them, and there come to rest, take some nourishment, and bring forth their little ones; while others of them lay their eggs there, in which new insects are inclosed. And finally, I suppose

[1] Joblot (1718), II. 38–39. Some of the "fishes" (*e.g. Colpoda*) are easily recognizable, but they had been seen and depicted by earlier observers.

[2] *Ibid.* II. 40.

that these same animals also drop their eggs and their young in the air wherein they disport themselves[1]."

It is unfortunate that Joblot gives so few details of his experiments. He says enough, however, to show that he had already conceived and carried out, in 1711, the very counterpart of those ingenious researches which become so celebrated, at a later date, in the hands of Needham and Spallanzani—and still later through the work of Pasteur and Tyndall. Joblot found—though in this he had been anticipated by Leeuwenhoek more than 30 years before—that "animalcules" are generated in an infusion even when the air is excluded. But he also discovered—and here apparently for the first time—that if the infusion be boiled, and then shut off from the air, no "animalcules" make their appearance. He even went a step further, and discovered that the boiled infusion, though "sterilized," has not lost its power of "generating life": for if it be exposed freely to the air once more, so that the "eggs" of "animalcules" can fall into it, it is still capable of generating abundantly. The idea of employing heat for sterilization in this manner does not appear to have occurred to anybody before Joblot[2], and his experiments—crude though they be, and quaintly chronicled—undoubtedly deserve mention whenever the more critical researches of Spallanzani and Pasteur are discussed. He was a "precursor" of both these greater men.

Joblot's pioneer researches in microbiology seem to have been overlooked by all save a few writers; and those who have called attention to his work—Fleck (1876), Cazeneuve (1893, 1894), Boyer (1894), Konarski (1895), Brocard (1905)—have unfortunately buried their own works so effectively that they have attracted but little attention. Full justice—perhaps more than justice—has, however, already been done to Joblot by Konarski (1895), in his masterly biographical essay. But on one important point I think Konarski's judgement is at fault: he gives Joblot credit for greater originality than he can fairly claim. According to his own statements[3] Joblot was studying "Infusoria" as early as 1680: that is to say, at the time when he had just been appointed to his post in Paris. He published his observations in 1718, and it appears that he had been engaged in making them during the whole of the 38 intervening years. Yet never once[4], in recording his researches on microscopic organisms, does he mention the name of Leeuwenhoek; and Konarski therefore concludes that Joblot, in all probability, was ignorant of Leeuwenhoek's work, and was,

[1] Joblot (1718), II. 45–46. It should be observed that this "hypothesis" was not original. It is closely similar to—and, in fact, very probably derived from—the views which had been put forward many years earlier by Leeuwenhoek, Huygens, John Harris, and others.

[2] The idea is commonly supposed to have emanated from Needham, at a later date (1745), and was extensively exploited by Spallanzani. Joblot, it may be noted, died before Spallanzani was born.

[3] Joblot (1718), II. 2.

[4] Only once does he mention his Dutch contemporary by name, and that in a quotation—in which he is called "Monsieur Leevuenhoec"—relating to the eyes of insects, and not to "Infusoria."

accordingly, himself an independent co-discoverer of the "Infusoria"—not a mere copyist of his great Dutch contemporary.

But such a supposition can hardly be defended seriously. As regards the publication of their results, it cannot be questioned that Leeuwenhoek was in the field 40 years before Joblot. Moreover, he had already published his discoveries relating to the "animalcules in waters" several years before 1680—the earliest date under which Joblot records any observations: and the discoveries were announced not only in the *Philosophical Transactions* in London but also in the corresponding French publication—the *Journal des Sçavans*—in Paris. It is hardly possible that Joblot could have remained in ignorance of Leeuwenhoek's discoveries, which created considerable stir at the time not only in scientific circles but also among the public. But furthermore, Joblot actually mentions[1] (in 1718) that Hartsoeker had shown him "at least thirty-eight years ago" the "Infusoria" in an infusion of pepper. Indeed, from the context it appears not improbable that this demonstration was the starting-point of Joblot's own observations. But we know that Hartsoeker, who, when he reached Paris, palmed off the "animalcules in pepper-water" as his own discovery, had himself learnt of them from Leeuwenhoek—their real discoverer—who had personally demonstrated some of his microscopic observations to Hartsoeker in Holland. Whether Joblot received his inspiration from Leeuwenhoek's writings or from Hartsoeker appears to be immaterial. Directly or indirectly he learnt of Leeuwenhoek's observations and discoveries, and then repeated his experiments. Other evidence in support of this view could easily be adduced from Joblot's own experiments[2], and from his intercourse with other investigators[3] who certainly knew and repeated others of Leeuwenhoek's observations—with acknowledgements. It is clear, to my mind, that Joblot was well acquainted with Leeuwenhoek's discoveries, though he never specifically mentions them: and whatever other inference we may draw from this regarding Joblot, we surely cannot conclude that his silence on the subject establishes his equality with Leeuwenhoek in the discovery of "Infusoria."

It is not to be denied, however, that Joblot added to our knowledge of the Protozoa and gave the earliest descriptions and figures of several species. He also invented a number of "new microscopes, both simple and compound," and did something to popularize their use and to demonstrate their possibilities as instruments of microbiological research. As a micrographer he has hitherto received less honour than certain other workers, who, though coming after him, did not surpass him in their powers of observation and investigation. In his experiments on spontaneous generation Joblot was, indeed, a pioneer, and in this connexion his name deserves to be recorded—after that of Leeuwenhoek—before those of Needham, Baker, Spallanzani, and Pasteur.

[1] Joblot (1718), II. 12–13.

[2] Many of them are obviously repetitions of similar experiments previously made by Leeuwenhoek. Konarski failed to notice this, apparently, because he was not acquainted at first-hand (as he says himself) with much of Leeuwenhoek's work.

[3] François Lamy, Louis de Puget, and others.

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C. G. EHRENBERG (1795—1876).

A BIOGRAPHICAL NOTE.

(PORTRAIT-PLATE XXV.)

BY CLIFFORD DOBELL, F.R.S.

CHRISTIAN GOTTFRIED EHRENBERG, one of the outstanding scientific figures of last century, was born on Easter Sunday, 19 April 1795, at Delitzsch. In this town—then in Saxony, but now in Prussia—his father, Johann Gottfried Ehrenberg, was a municipal official, who was thrice married; his second wife being the mother of Christian Gottfried, who was the eldest son. His mother's maiden name was Christiane Dorothea Becker, and she was the daughter of the innkeeper of "The Red Lion" in Delitzsch. The forefathers of these Saxon Ehrenbergs were mostly millers, and they appear to have been unrelated to the noble family of the same name.

Young Gottfried went to school at Pforta (1809–1815), where he received a sound classical education and spent his leisure in studying natural history, especially botany. In 1815, when he was just twenty, his father sent him to Leipzig University to study theology; but he soon abandoned this uncongenial subject and turned his attention to medicine instead. He left Leipzig in 1817, however, and entered the University of Berlin, where he continued his medical studies and acquired considerable proficiency in botany and zoology. When he took his medical degree, in 1819, his inaugural dissertation dealt with the Fungi growing about Berlin.

In 1820 a Prussian officer, General von Minutoli, conducted, with the support of his Government, an archaeological excursion to Egypt: and to his party Ehrenberg and his friend W. F. Hemprich, a young botanist, were attached as naturalists. The expedition was a failure. It is generally referred to as "ill-fated," but it seems to have been equally ill-organized; and the published accounts of its doings would be comic were they not often so tragic. Ehrenberg himself has said that their caravan "was more like a field hospital than a company of scientific investigators." It soon broke down completely, but Ehrenberg and Hemprich were able to continue their travels. They journeyed through Egypt, Libya, Nubia, Sinai, and Arabia, and explored especially the Red Sea. After encountering an incredible number of misfortunes and difficulties, they finally reached the coast of Abyssinia, where Hemprich died of fever. Ehrenberg, seriously ill himself, then gave up all hope of being able to continue further, and set out for home—reaching Berlin

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Separate copies may be obtained from the University Press, Cambridge

once more in 1826. With the exception of a few minor excursions abroad—the most important being a journey through Russia and Siberia with his friend and patron Alexander von Humboldt in 1829—Ehrenberg remained at the University of Berlin for the rest of his life. During the greater part of this time—approximately fifty years—he endeavoured to work out the scientific results of his travels; but the immense mass of botanical and zoological specimens and observations which he and Hemprich had collected became a millstone to his neck, and, despite the help which he received, he died with this, the great work of his life, still unfinished. Under the title *Symbolae Physicae* instalments of it began to appear in 1828, and continued intermittently until the beginning of the present century (1900): but the publication is still a "torso"—as Ehrenberg himself styled it—and is likely ever to remain so.

At Berlin Ehrenberg was made Extraordinary Professor in 1827, and Professor in 1839: but although he belonged to the medical faculty he never took much interest in medicine, and devoted his attention chiefly to the study of microscopic organisms. Such lectures and demonstrations as he gave attracted zoologists, botanists, and geologists, but for the most part they were entirely devoid of medical interest.

Soon after he had settled down in Berlin (1831) Ehrenberg married Julie Rose, a daughter of the Danish consul at Wismar. After her death he subsequently married (1852) Lina Friccius, a niece of Mitscherlich, the chemist. By his first wife he had five surviving children—a son and four daughters. In his old age his youngest daughter, Clara, was his constant help and companion. She mounted his preparations, wrote out his notes, and even, when both his eyes were afflicted with cataract, made his microscopic observations for him; and finally she tended him in his last illness and buried him. He died peacefully, aged a little over 81, on 27 June 1876—full of years and loaded with honours. But he had had practically no pupils and so he left no school behind him. "He was essentially a man who did his work alone."

It is said that Ehrenberg, at the time of his death, had been elected a member of no less than seventy learned societies. He had also received many decorations, orders, and prizes, in recognition of his scientific achievements. The Berlin Academy took him to her bosom when he was still a young man (1827): the Royal Society elected him a Foreign Member ten years later (1837): and in 1860 even the Institut de France honoured him with membership. He knew most of the scientific workers and notable people of his day, and had a wide circle of friends and admirers—counterbalanced, it is true, by a considerable band of enemies and critics who cordially detested himself and all his works. His early life was a series of misfortunes, but these were fully compensated, in a material sense, by the honours lavished upon him later. He enjoyed his laurels in his lifetime.

Ehrenberg was not a parasitologist, and his claim to notice in the pages of *Parasitology* rests upon his contributions to our knowledge of all kinds of

microscopic organisms—more especially the Protozoa and Bacteria. He was at heart—though he hated to be told so—a collector and systematist, and he largely helped to found Bacteriology and Protozoology by identifying, naming, and cataloguing all the microscopic creatures—animals and plants, both living and fossil—that his industry could discover. He was, besides, a diligent reader, conversant with Hebrew and Arabic as well as with Greek and Latin and many modern European tongues, and he assiduously garnered everything that had ever been written by his predecessors on any subject which he treated. He was also so copious a writer that he deserves the epithet πολυγραφώτατος which Diogenes Laertius applies to Epicurus: for while the latter is but credited with having covered 300 cylinders with his writings, those of Ehrenberg amount to something nearer 400, and he had a fondness for extra-large folios and quartos. It will thus be evident that no detailed account of his various botanical, zoological, anthropological, histological, geological, geographical, meteorological, archaeological, and other publications can be attempted here. A few words on his contributions to protozoology and bacteriology must suffice.

When Ehrenberg wrote, "protozoology" and "bacteriology" were unknown words. He classified all the Protozoa and Bacteria known to him in one grand group—the "Infusoria"—along with the Rotifers, the Diatoms, and the Desmids. All of these he regarded as animals. Wherever he went he collected these "Infusoria" and studied them and drew them; then later he named them, catalogued them, and wrote about them. He looked for and found them in ponds, rivers, seas, lakes, earths, rocks, and in the air. When he was aged 43, he collected everything he knew, and all that he could find that had ever before been written about them, into one enormous book entitled *The Infusoria considered as Perfect Organisms*—a large folio of 548 pages, accompanied by an atlas of 64 coloured plates. This truly monumental work is a milestone, rather than a foundation stone, in the history of protistology; but it did for microscopic organisms what the *Systema Naturae* of Linnaeus did for the large animals and plants. It contains an immense number of accurate records and observations imbedded in a matrix of mistakes, so that even now it is hard to assess its true value. Everyone who studies protozoa, bacteria, desmids, diatoms, or rotifers, must consult it: none of those who do, however, will be able to allow the entire correctness of what he there finds written.

It is impossible to review such a work now, but to give a sample of the information contained in it I may perhaps enlarge a little here upon the term "Polygastrica"—a word which constantly appears in its pages. This term was coined by Ehrenberg to denote the organisms now comprised in the Mastigophora, Rhizopoda, Ciliata, Acinetaria, Bacteria, Bacillarieae, and Desmidiaceae. All these he regarded as animals, and chiefly for the reason—implied in the word Polygastrica—that they are provided with numerous stomachs. The presence of these organs is demonstrated by feeding the

animals with particles of carmine or indigo. Since they contain stomachs, they must also have a mouth, an intestine, an anus, and to make them complete a nervous system, muscles, excretory and generative organs, and so on. All these "animals" are, in fact, organized in the same sort of way as the large ones—they are complete and perfect (*vollkommen*). It is easy to understand Ehrenberg's observations and interpretations when he is dealing with an animal like a ciliate. The "stomachs" were food-vacuoles, and the behaviour of the indigo particles was accurately observed—only the interpretation of the phenomena being wrong. But when other organs or inclusions in these animals are interpreted as ovaries, testes, gall-bladders, and so forth, it is not always so easy to follow his argument; and when similar things are then described in a diatom, the modern reader may even find difficulty in taking the observations themselves seriously. Yet this is the sort of information which Ehrenberg gives us about an "infusorian." Under a well-chosen name he generally records some accurate observations, an occasional experiment, many misinterpretations, and gives a picture which often affords a key to his words; while the whole is neatly rounded off with valuable references to the works of others—albeit his judgement in the interpretation of their findings is frequently at fault. The chief group of parasitic protozoa —the Sporozoa—was practically unknown to Ehrenberg. Had he met with them he would doubtless have found "stomachs" in them too, for he refused to entertain the notion that any animal could absorb nourishment "like a sponge or blotting-paper." To the end of his life, moreover, he continued to view with disfavour the new-fangled idea that "microbes" can cause diseases.

The fossil micro-organisms were described—with many other things—in a second huge folio on *Microgeology* published some years later. This is a worthy companion to the earlier work. Among Ehrenberg's other works on kindred subjects there is only space to mention here the series of papers dealing with organisms borne by the air. The observations were collected and summarized in 1871, and undoubtedly had an influence in the early days of bacteriology. Yet Ehrenberg's name is now chiefly quoted by systematists. He named hundreds of animals and plants, and not a few were named after him: his own name was given also to a hill, a cape, a group of islands, and a mineral (Ehrenbergite), though not all of these designations have survived. Nevertheless, one has only to recall such familiar Ehrenbergian genera as *Euglena*, *Bodo*, *Chlamydomonas*, *Bacterium*, *Spirillum*, *Spirochaeta*, *Glaucoma*, and *Frontonia*, to realize that he left his mark imperishably on protozoology and bacteriology.

It is now clear that Ehrenberg's great work on "Infusoria" was published unseasonably. In almost the same year the cell doctrine of Schleiden and Schwann made its appearance, to be soon followed by the revival of the theory of organic evolution. Ehrenberg's views were incompatible with either of these theories—both of which he ignored as far as possible, though they, with the accurate observations of Dujardin and others, soon brought about

the downfall of his favourite dogmas. Nevertheless, he stuck to his beliefs, and even at the end of his life spoke confidently of having "proved" (*scharf erwiesen*) that diatoms are animals possessing stomachs. Underlying all his mistakes in matters of detail, however, there is a solid substratum of truth: and it is probable that his general conception of the "perfect" organization of the Protozoa is not dead but only dormant, and will be heard of again.

Our Plate shows Ehrenberg when he was somewhat over 60 years of age. In reality his face was pitted by small-pox, from which he suffered when three years old. His benevolent and almost childlike features, conjoined with his very small stature—he tells us himself that he was "not more than five feet high"—contrast strikingly with the remarkable industry and erudition which his life and writings reveal. It is difficult to realize that this apparently genial little old gentleman composed so many large and learned treatises: and still harder is it to picture him indulging in embittered arguments—as he undoubtedly did at times—and tenaciously defending positions which now appear ridiculously untenable. But as Bütschli, who had a singularly competent knowledge of his work, has well observed: "It was not one of Ehrenberg's peculiarities to relinquish a notion when he had once conceived it, especially if it met with contradiction." When still a young man he bought a microscope—no doubt at that time a very excellent one: but though microscopes and lenses underwent vast improvements during his lifetime, he consistently refused to give up this old instrument. It had been, in his opinion, the best microscope; consequently, it remained ever the best microscope: and there can be little doubt that many of his mistakes arose through his faith in this primitive machine. He generally stood up when he used it, as he considered sitting to be a less suitable posture for making the most accurate observations. He also favoured dress clothes for everyday wear, and a top hat. Queen Victoria, who saw him at Cambridge in 1847—when he received an honorary M.A. degree from the hands of the Prince Consort—has left it on record that he then appeared "very odd and very German-looking[1]."

Besides being a man of science Ehrenberg was also something of a poet, and a draughtsman of no mean ability, as the thousands of figures illustrating his works amply testify. His pictures are, indeed, often superior to his descriptions; and the statement which has been made that many of his figures of protozoa are worthy of a place in a modern text-book, contains an unmerited—if unintended—censure.

As may well be imagined even from the foregoing imperfect outline, a man of such remarkable parts has not lacked biographers. His daughter Clara collected a mass of information bearing upon his life and work, but did not live to publish the results of her devoted labours. Nevertheless, her materials were preserved and came later into the hands of a kinsman—Max Laue—

[1] It is only fair to say, however, that he had lost his luggage on the way to Cambridge, and arrived in a very travel-stained condition.

who published an authoritative and fully documented biography of Ehrenberg in 1895. This book, which contains a full list of Ehrenberg's writings and a fine portrait, describes his life in considerable detail, and has been, with the less important works of Hanstein (the botanist, Ehrenberg's son-in-law) and Hesse, my main source of information for the foregoing article; though my estimate of Ehrenberg as a microbiologist is based upon personal study of his works—which are far too numerous to cite here, and are, moreover, full of biographic material—and not upon the eulogies of his admirers or the defamations of his detractors.

The chief works referred to in the foregoing article are:

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—— (1854). *Mikrogeologie. Das Erden und Felsen schaffende Wirken des unsichtbar kleinen selbständigen Lebens auf der Erde.* Folio. Leipzig.

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—— (1871). Uebersicht der seit 1847 fortgesetzten Untersuchungen über das von der Atmosphäre unsichtbar getragene reiche organische Leben. *Ibid.*, No. 1, pp. 1–150.

HANSTEIN, J. (1877). *Christian Gottfried Ehrenberg. Ein Tagwerk auf dem Felde der Naturforschung des neunzehnten Jahrhunderts.* 8°. Bonn. [A shorter biography by the same author will be found in *Allg. Deutsche Biogr.*, Bd. v, pp. 701–11. 1877.]

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SPECIATION AND HOST RELATIONSHIPS OF PARASITES[1].

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In recent years the speciation of parasites as related to their host relationships has presented some of the most interesting and at the same time most difficult problems in the field of parasitology. These problems are by no means of purely academic interest but are of prime importance in connection with the control and prophylaxis of a number of important infections of man and domestic animals.

Two diverse tendencies are displayed among parasitologists at the present time with regard to these questions of speciation and host relationships of parasites. One tendency is to look upon parasites living in two different hosts, or in two different habitats in or on a single host, and more or less constantly showing slight morphological differences, as distinct species until there is some positive evidence for considering them identical; the other tendency is to consider such parasites as identical until definitely shown to be distinct, thus shifting the burden of proof from one side to the other. Until recently the former tendency has been more pronounced, and much emphasis has been laid on slight morphological differences in parasites living in different hosts or under diverse conditions. However, with the growing evidence for the fact that when a parasite is transferred to a different host it may undergo certain morphological changes which are more or less constant so long as it remains in or on the new host, together with increasing evidence that parasites in the course of a few generations increase their adaptability to a given host, at the same time possibly losing adaptability to other hosts, the tide of opinion has slowly but steadily been turning in the opposite direction. In some instances a more critical and careful re-examination of closely related "species" has shown that the supposed constant morphological differences either do not exist or have been greatly exaggerated.

Before entering into a discussion of the mechanism of speciation and adaptation to hosts on the part of parasites, it would be advantageous to discuss, briefly, some of the conspicuous illustrations of groups of closely related parasites inhabiting different hosts, which have formerly been, and in many instances still are, bones of contention between those who would make of them several species, and those who would combine them into a single

[1] Presented before the Helminthological Society of Washington Mar. 17, 1923.

species with a number of varieties or physiological races. Such illustrations can be found among all three of the principal divisions of parasites—the arthropods, the helminths, and the protozoa.

One of the most interesting and instructive examples among the arthropods is the case of the mites of the genus *Sarcoptes* which cause scabies, itch, or mange in man and various animals. These mites were divided by Berlese into no less than fifteen different species, and by Canestrini and Kramer into eighteen species, four of which, however, are considered doubtful. On the other hand there are many parasitologists who prefer to accept only a single species of *Sarcoptes*, *S. scabiei*, "which may give rise to different races or castes by living in the skin of man or mammals, but can pass from one host to the other" (Fantham, Stephens and Theobald, 1916). As has been pointed out by Munro (1919) and again by Warburton (1920) the cause for the confusion in this case has been very largely a lack of accurate knowledge. Many of the descriptions of "species" of *Sarcoptes* date back to 60 or 70 years ago when microscopes and technique were inadequate for proper study of organisms which vary in such minute details as do these mites, and in many instances the descriptions are based on inadequate material. Recently a very careful examination and comparison of the itch mites from the horse and from man has been made by Buxton (1921) in which no constant characters were found which could be used in differentiating the species.

The actual morphological differences which occur *on the average* between "species" of *Sarcoptes* living on different hosts are in size, proportion between length and breadth, pigmentation, cuticular markings, and chaetotaxy. It is significant that these are precisely the kind of differences which have for decades been used in separating the two kinds of human lice, the head louse and the body louse. Recently Nuttall (1919) has shown that there are actually no constant differences between these two kinds of lice, and Keilin and Nuttall (1919) and others, have demonstrated that when head lice are raised experimentally under conditions which are favourable for the development of the body louse, they lose all their own distinctive morphological features, and acquire all the morphological characters of the body louse after four or more generations. It is interesting to note also, in this connection, that in recent publications Fahrenholz, a noted German authority on lice, has described four subspecies of body lice and three of head lice from various races of man, and four species from apes and monkeys, all based on what are probably inaccurate observations or on characters which Nuttall in his extensive research has shown to be useless for diagnosis of species or subspecies, *e.g.* size, proportions and pigmentation.

A situation quite comparable with that existing in *Sarcoptes* and *Pediculus* is to be found among the "species" or varieties of *Chorioptes* and *Psoroptes*, and the hair follicle mites of the genus *Demodex*.

A number of similar instances exist among the helminths. A good example is the case of the dwarf tapeworm of man, *Hymenolepis nana*, and its counter-

part found in rats and mice. There are slight average morphological differences between the human and rodent forms of this parasite, but not greater than might reasonably be expected in a single species living in different hosts; *Syngamus trachealis*, the gape worm, for instance, when it develops in turkeys reaches a length almost double that attained in chickens. Furthermore, some of the alleged differences between the human and rodent forms of *Hymenolepis nana* are due, as Baylis (1922) has pointed out, to the confusion of two quite distinct species in rats. A number of attempts at cross-infections with the dwarf tapeworm between man and rodents have been made with inconclusive results. Grassi succeeded in infecting one out of six boys in Italy by feeding them ova from a rat parasite, but in a place where the human infection is of common occurrence and where no adequate precautions were taken to prevent infection from other sources this cannot be considered conclusive evidence. Recently Saheki (quoted by Yoshida (1921)) claims to have infected rats and mice, as well as man, by feeding eggs from a human infection. Attempts to infect rats with ova from human parasites have been made by Joyeux (1920), Chandler (1922) and others, but with negative results. Although significant in showing that these infections are not *readily* transmitted from one host to the other, they are by no means extensive enough to be conclusive.

Other instances of related helminths from different hosts which may prove to be acclimatised races of a single species may be found among many other genera of nematodes, cestodes and trematodes. Of interest from the point of view of the parasites of man or domestic animals may be mentioned *Ascaris*, *Belascaris* and *Trichuris* among Nematodes, *Taenia* and *Cittotaenia* among cestodes, and *Paragonimus* and *Opisthorchis* among trematodes.

It is among the Protozoa that the most numerous instances of disputed identity, and the most difficult problems of host relationships, are to be found. Probably the group of Protozoa which best exemplifies this is the *Leishmania-Herpetomonas-Trypanosoma* group of flagellates. These organisms have for decades presented a problem bristling with inaccurate observations, insufficient knowledge, erroneous deductions and unwarranted conclusions. Probably no other group of parasites has led to so much diversity of opinion and so much heated argument. Many of the problems involved will probably not be definitely settled for many years to come.

There is at present not very much doubt but that this entire group of flagellates may be considered as having evolved from simple flagellate parasites of the gut of invertebrates. Many are, so far as is known at present, still confined to their invertebrate hosts, others alternate in their life cycles between vertebrate and invertebrate hosts, while still others are apparently quite independent of their presumably original invertebrate hosts and are, facultatively at least, exclusively vertebrate parasites.

The confusion which exists at present concerning the relationships of various gut flagellates of invertebrates, more of which are constantly being described, and the *Leishmania* and trypanosome infections of vertebrates, is

becoming worse rather than better. Are the numerous invertebrate flagellates described from different hosts really distinct species or are many of them merely races or castes of a single species, slightly modified in response to the differences in the environment in the intestines of the different host species? The latter suggestion has been made by Mackinnon (1910) after a careful study of the herpetomonads of a number of species of non-biting flies. A further question arises as to the relationships of the parasites causing the various forms of dermal leishmaniasis in vertebrates in different parts of the world. Are these infections due to one, two or numerous species of flagellates? Are these flagellates specifically distinct from the forms which are supposed to be confined to invertebrate hosts, or may they be regarded as strains or races of the latter which have acclimatised themselves to certain kinds of vertebrate blood as the result of exposure to it in the gut of the invertebrate host? Can a line be drawn between a genus *Leishmania*, which includes those forms existing as herpetomonads in invertebrates and as *Leishmania* forms in vertebrates, and a genus *Herpetomonas* (or *Leptomonas*) normally confined to the invertebrate? There is no doubt a sharp distinction, in life history at least, between such a form as *Herpetomonas muscae-domesticae* of house flies, and *Leishmania donovani* in human beings. But when we recall that in many parts of the world cases of dermal leishmaniasis sporadically occur, said to result from the bites of tabanids, simuliids, chironomids or ticks, and that typical vertebrate species, such as *Leishmania donovani* and *L. tropica*, undergo developmental cycles in the gut of bedbugs and *Phlebotomus*, and that herpetomonads develop in the gut of insect-eating lizards, one can reasonably wonder whether there is any valid distinction between vertebrate-infecting and non-vertebrate-infecting groups of species. The line of distinction would fade completely if the work of Laveran and Franchini and Fantham and Porter in artificially infecting mammals and birds by inoculating them with purely insect flagellates should be substantiated, but their results have not been confirmed by others. (See Hoare 1921, and Glaser 1922.)

The situation among the trypanosomes is somewhat better understood than is the case among other representatives of this group of flagellates, because many of them have been isolated and studied in pure culture and by animal inoculation for many years, the various species or strains being determined by cross immunity reactions and pathogenicity. Such a proceeding has not yet been extensively employed in the study of the *Leishmaniae* and herpetomonads.

Among the African polymorphic trypanosomes of mammals numerous species or strains have been obtained from various hosts, both vertebrate and invertebrate, and have been differentiated more on the basis of their biological than their morphological characteristics. But the doubtful value of this differentiation is well expressed by Duke (1921) in the following words, "One of the most striking results of the European work on the polymorphic mammalian trypanosomes of Africa has been the revelation that, in the course of years,

the laboratory strains of these organisms may undergo great changes in morphology, in virulence, and in their immunity reactions. A strain of trypanosomes may thus become, after some years of laboratory upkeep, very different from what it was originally, when freshly isolated from its natural host; and this has led to the splitting up of "species" and consequent confusion in classification." That a similar splitting up might take place in nature under circumstances where direct rather than cyclical transmission by tsetse flies takes place, no one would doubt, but Duke has adduced some evidence that even cyclically transmitted trypanosomes in nature, persistently transmitted to different groups of vertebrate hosts, tend to differentiate into strains comparable with the asexually propagated laboratory strains.

A very striking case of what is thought by many parasitologists to be hereditary adaptation of a species to a new host is furnished by the sudden appearance in Rhodesia and Nyasaland of a new form of sleeping sickness caused by a trypanosome indistinguishable from *Trypanosoma brucei*, the cause of nagana in domestic animals, except by its pathogenicity for man. The prevalent opinion is that this trypanosome, known as *T. rhodesiense*, is merely a race of *T. brucei* which by some slight physiological change, possibly brought about by more frequent exposure to human blood in the gut of the fly, has become capable of development in man. Duke goes farther and advances convincing arguments, supported by strong circumstantial evidence, that all the polymorphic trypanosomes of Africa are varieties of a single species. "These strains," says Duke (1921), "are not immutable but variable, and are determined by the environment in which the species lives; they are not constant under varying external conditions, but each different strain is dependent upon, and is produced as a response to, a particular environment."

A very interesting comparable situation exists in tropical America. It has been demonstrated that in a number of parts of Brazil and possibly also in the northern parts of Argentine, human beings are commonly affected by a pathogenic trypanosome, *T. cruzi*, which ordinarily utilises a Reduviid bug, *Triatoma megista*, as an intermediate host, but which has been shown to be capable of development in other species of *Triatoma*, and also in other kinds of Rhynchota and in certain ticks. *Triatoma megista* habitually lives in human habitations, feeding on the occupants at night, therefore the flagellates harboured by them would have abundant opportunity for acclimatisation to human blood in consequence of repeated exposure to it. In many parts of tropical and subtropical America from Argentine to the Southwestern United States, trypanosomes resembling *T. cruzi*, and in some instances morphologically indistinguishable from this species, have been found to be harboured by various species of *Triatoma*, although in much of this territory no human trypanosomiasis is known to occur. It is a significant fact that in these regions the *Triatomae* do not habitually live in association with human beings and only incidentally attack man. They normally feed on various species of

rodents, armadillos, or other animals. It seems to the writer not only possible but probable that the case here is directly comparable with that among the polymorphic African trypanosomes, and that these trypanosomes all represent strains or races of a single species more or less modified in response to differences in environment. The wide adaptability of *T. cruzi* to such different mammalian hosts as armadillos, dogs, rodents and primates parallels that found in the *T. brucei* group in Africa, and just as the variation in habits of tsetse flies and availability of different kinds of food animals in different parts of Africa results, apparently, in the differentiation of strains of the polymorphic trypanosome, so in America the habits of different species of *Triatoma* and allied bugs and availability of different hosts might readily be conceived of as leading to a similar differentiation of strains of *T. cruzi*, which would then be found to differ in morphological details and pathogenicity.

Looking outside this confusing group of flagellates, and turning our attention for a moment to the intestinal protozoan parasites of vertebrates, we find many similar conditions. The probability of some of the numerous species of flagellates described from various species of Amphibia being slightly modified representatives of a single species has been pointed out by Alexeieff (1909). Among the intestinal flagellates affecting human beings, *Trichomonas* serves as a good example.

In man *Trichomonas* occurs in the intestine, the vagina and the mouth, and in each situation shows certain morphological peculiarities. Moreover, investigators differ in their descriptions of the organism in different series of examinations or in different parts of the world. Thus varieties or "species" have been described having three, four and five flagella, and these have even been placed in distinct genera. Faust (1921), from a review of the work of other investigators, and from an examination of his own material, comes to the conclusion that there is but one species of human intestinal *Trichomonas*, in which the number of anterior flagella varies from three to five but tends to be constant in particular races. Such a racial variation would be comparable, perhaps, with that of the races of *Entamoeba histolytica*, which differ as regards the size of their cysts. The forms from the intestine, vagina and mouth are commonly regarded as distinct species, since they show demonstrable morphological differences. A physiological difference is evident since *Trichomonas vaginalis* lives in vaginal mucus having an acid reaction, whereas *T. hominis* is exposed to an alkaline medium in its habitat in the intestine. But until it has been experimentally shown that *Trichomonas hominis* cannot adapt itself under favourable conditions to life in the mouth or the vagina, and when thus adapted does not show morphological modifications, the possibility of the three human types of *Trichomonas* being races of a single species cannot be excluded. Certainly much more work needs to be done on individual or racial variation in such Protozoa as *Trichomonas* before one can feel confident that the differences exhibited by this parasite under varying environmental conditions are of specific value.

The *Trichomonas* problem is further complicated by the occurrence of very slightly different forms of the parasite in rats, mice, guinea-pigs and hogs. Although usually considered as distinct species, there is no conclusive evidence that the *Trichomonas* of man, rodents and hog may not be varieties of a single species adapted to live in different hosts, as the various forms found in man may be races adapted to different habitats in a single host. Essentially similar cases of closely related parasites are to be found in *Giardia* and *Entamoeba* and in many other genera.

It is interesting to note that a condition parallel with some of the cases mentioned above exists among the problematical *Rickettsia*-like organisms which occur in the gut of insects, and which, like the flagellate intestinal parasites of invertebrates, show adaptability to life in vertebrate animals in some instances but not in others. Organisms of this type are believed to be the causative agents of typhus fever, trench fever, Rocky Mountain Spotted fever, and possibly the Japanese disease, Kedani or Tsutsugamushi. The *Rickettsia*-like organisms associated with these diseases are found in abundance in the arthropod vectors, but organisms of the same type are found also in fleas, sheep-ticks, bed-bugs, and Mallophaga. Furthermore, in body lice which apparently have had no opportunity to become infected from either typhus or trench fever patients, these organisms have been found. It has been suggested that there is probably a third species found in lice which is specific to the insects and which, like those found in other insects, some of which are not bloodsuckers, is, so far as known, incapable of development in vertebrate animals, and is therefore non-pathogenic.

It should be recalled, however, that although typhus has been known for many centuries, trench fever was unknown before the outbreak of the European War. It gradually became recognised as a distinct disease during the latter part of 1915, and by the end of 1916 was observed on all the war fronts in Europe. It is stated that during the last two years of the war no other infective disease occasioned so much sickness among the troops in France. Various hypotheses have been advanced to account for the sudden appearance and almost unprecedented development of a previously unrecognised disease. Some writers have endeavoured to identify it with previous epidemics, others maintain that it was present in Europe before the war but was mistaken for various other diseases which it may more or less simulate. Still others have supposed that it is some obscure exotic disease, introduced by the colonial troops, which on account of the favourable war-time conditions developed into an epidemic of unprecedented proportions. Still others believe that it is actually a new disease.

Whether or not trench fever existed before the war, certainly it was at best an obscure, little known infection of local distribution, both as regards time and space. To the writer the most probable explanation is that the sudden appearance of trench fever was due to the development of a strain of the normally harmless *Rickettsia pediculi* of lice which was capable of

multiplication in human blood, and that this physiologically new strain of the organism, under the ideal epidemiological conditions afforded by the war, became firmly established and more readily infectious by repeated passage through human beings, and was then spread all over Europe by the movement of troops and the abnormal abundance of lice. Had such a strain of the organism been developed under normal conditions, without the opportunity for quick repeated passages through individuals who were unaccustomed to lice and who could not possibly have developed any immunity against it by repeated inoculation with small numbers of the harmless strains, it might readily be conceived that it would not have succeeded in obtaining a foothold, or even if it had done so, would have died out after a temporary local epidemic.

Such instances of the sudden appearance of new human diseases or of the sporadic outbreak of others as are to be observed in the case of trench fever, Rhodesian sleeping sickness, Norwegian itch and dermal leishmaniasis seem to the writer to be excellent examples of the instability of infective diseases. As has been very clearly pointed out by Lloyd (1912), if infective disease is to be regarded as the interaction of particular organisms, there must be an evolution of disease, as there is an evolution of the organisms concerned. Appreciable changes both as regards morphology and habits are known to occur suddenly and sporadically in the succession of organisms. Whether we consider them as mutations in the ordinary sense of the word, *i.e.* as the occurrence of entirely new characteristics, or as outcroppings of characteristics contained, but usually concealed, in the parent species, is immaterial from a practical standpoint.

It is obvious from the examples discussed in the preceding pages that our knowledge concerning some of the most important parasites is in a very confused state and that a great deal of well controlled experimentation is required before the vexed problems of speciation and host relationships in parasites can finally be solved. Some of the suggestions made above lie clearly in the field of speculation, but they may serve as working hypotheses for future work, and even though eventually proved false, may in the meantime fulfil a useful purpose.

In looking over the entire range of parasites—arthropods, helminths, and protozoa—one cannot help but be impressed by the recurrence of the same problems in every group. In mites and lice, in nematodes and tapeworms, in trypanosomes and trichomonads, in the borderland *Rickettsia*-like organisms and, it need hardly be pointed out, in spirochaetes and bacteria, we find closely related forms living in different hosts, or in different habitats, differing from each other, if at all, in slight morphological details, and varying in infectivity for different hosts. One can hardly escape the conviction that in all of these groups the same or similar factors are at work. It is probable that all parasites, whether arthropod, helminth or protozoan, undergo slight physiological changes in order to become acclimatised to different kinds of environ-

ment. These physiological changes may have to be present before the new environment can be invaded at all or they may be developed in response to the altered environment after exposure to it. Physiological changes inevitably go hand in hand with morphological changes, although the latter may or may not be obvious.

In considering the possible mechanism by which these physiological and morphological alterations are brought about, and by which they become progressively more fixed and more distinct in the course of continued exposure, generation after generation, to altered environmental conditions, it is necessary at the outset to distinguish between somatic acclimatisation and genetic modification. The instances mentioned above among metazoan parasites of modification on exposure to altered environment are clearly genetic in nature, since they occur among sexually reproducing organisms. Among Protozoa the possibility exists that some of the modifications displayed in different environments may be due to non-heritable somatic alterations. Trypanosomes, for instance, can be made highly resistant to drugs by exposure to gradually increasing doses. Gonder (1911) has shown that this drug resistance, although it remains constant through thousands of asexual generations during the passage by inoculation from rat to rat, is entirely lost after passage through the intermediate host, where some process analogous to sexual reproduction presumably takes place. It seems hardly reasonable to believe that the drug resistance developed by asexually multiplying trypanosomes is due to natural selection and isolation of a naturally immune race, although some races might acquire immunity more readily than others. Adaptation to the blood of a new host might at first sight be considered comparable to this drug acclimatisation, especially in view of the fact that the drugs act in conjunction with the serum, but there is one important difference. The drug resistance is built up by a gradual process of immunisation, whereas resistance to a new serum or other body fluid cannot be acquired by numerous steps but must be attained at a single jump. Just as among trypanosomes exposed to increasing doses of drugs there may very likely be some races which do not have the power of acquiring resistance, and are therefore weeded out early in the immunisation process, so among protozoa exposed to new body fluids, there may likewise be races which are less adaptable than others, and therefore eliminated. In the case of drugs, we presumably have somatic acclimatisation acting upon selected genetic races which have the power of acclimatisation, while on exposure to a new kind of body fluid the process of adaptation depends largely upon the initial selection of genetic races which are capable of withstanding the change.

There are, in fact, a number of good reasons for believing that the differentiation of physiologically distinct races of protozoa is due to a process of selection of genetic races rather than to somatic acclimatisation. In the first place it has already been pointed out that the problems of host relationships are so similar in all three groups of parasitic organisms that little room is left

for doubt that factors are at work among Protozoa similar to those among the helminths and arthropods. In the second place, although sexual processes have not been recognised in the majority of parasitic protozoa, yet a process equivalent to sexual reproduction, *e.g.* autogamy or endomixis, almost certainly occurs. In the intestinal protozoa in which no intermediate host is involved, this process presumably occurs just before or just after cyst formation, whereas in the trypanosomes it presumably occurs in the invertebrate host. If this is correct then the hostal races of protozoan parasites as well as of metazoan parasites are to be regarded as genetic in nature. Most instances of the development of differentiated strains of asexually multiplying protozoa are undoubtedly due to genetic differences, since such strains are distinguishable from each other, even when environmental conditions are varied. Jennings (1909, 1911) for instance, was able to obtain a whole series of strains of *Paramoecium*, differing in size, each of which would breed true asexually, though, owing to their hybrid nature, their purity was broken up by effective recombinations of characters where conjugation occurred. Erdmann (1920) has demonstrated that a similar breaking up occurs as the result of endomixis. Recently Taliaferro (1921) has demonstrated the presence of genetically distinct races in *Trypanosoma lewisi* which differ in size and maintain a mean with a very low coefficient of variation throughout numerous transfers through rats. Yet when these races are passed through the flea which acts as an intermediate host the coefficient of variation is invariably significantly increased, showing that the line is broken up during the passage through the flea, undoubtedly as the result of some nuclear reorganisation similar in its effect to sexual reproduction. Erdmann, in her experiments with *Paramoecium*, found that although races are broken up by endomixis, there is a tendency for those parthenogenetically produced races showing the same size characteristics as the parent stock to survive if environmental conditions are kept uniform, due to survival of the fittest. After previous occurrences of endomixis the race best adapted to the particular environment has survived, and is likely to continue to do so in spite of the temporary appearance of other races developed at each period of endomixis.

It appears to the writer highly probable that a similar explanation may account for "hostal" races in parasites. Most investigators are now convinced that the majority of wild, *i.e.* natural, races of animals are hybrid in nature, and that from them a number of lines or races differing in morphological and physiological characteristics may be isolated. If there is no selective action of the environment, all of these lines or races would tend to appear more or less regularly after each occurrence of sexual reproduction or nuclear reorganisation. If there *is* a selective action of the environment, on the other hand, and some races are better adapted than others for survival, then the less adaptable races would soon tend to die out and the more adaptable ones would persist, a species under given environmental conditions showing, therefore, a considerable degree of uniformity. On the other hand, if the environ-

ment were altered, there is every reason to suppose that another race might be more adaptable than the parent one, and this would account for the morphological and physiological differences exhibited by parasites found in different hosts or habitats. Itch mites living in the skin of man, for instance, show certain average differences from those found in horses or pigs due to the fact that the races which are best adapted to life in human skin are not the same as those which are best adapted to life in the skin of the other animals. The less adaptable races in each case either die out, sooner or later, or fail to reproduce. A similar explanation would account for the variations found in such a protozoan parasite as *Trichomonas*. Environmental conditions in the human mouth, vagina and intestine, and in the intestines of man, rats and guinea-pigs, presumably differ sufficiently to exert a selective action on races of the parasite occurring in them. As a result of continued inbreeding and selection it is possible that some potential races would be eliminated entirely and the combination of favourable characters become more and more frequent.

It is possible that not all modifications of parasites in different habitats are due to this process of segregation and survival of favoured genetic races, since some may be due to pure somatic acclimatisation and others to "induction" or "preinduction" effects such as those demonstrated by Whitney in rotifers, by Woltereck in *Daphnia* and by Sumner, possibly, in mice. Nevertheless, the segregation of adaptable races stands out as the most probable and most frequent factor involved in the formation of "hostal" races. And whether this explanation be accepted or not, it seems to the writer that the known facts favour an extensive recognition of the widespread impurity of species in nature, of the instability of the physiology and morphology of parasites as well as of other animals, and of the consequent inconstancy of infectivity and pathogenicity, and that for this reason it would be wiser, for the present, to await some positive evidence of the multiplicity of species of closely related parasites living under different environmental conditions before elevating them to the rank of species and giving them specific names. On the other hand, the tendency to lump together parasites from different hosts or different habitats without nomenclatural distinction is an error the seriousness of which cannot be too strongly emphasized. Many scientific observations lose much if not all of their value from the fact that the observer was not sufficiently accurate in defining the form or forms with which he was dealing.

Davis (1922) has recently called attention to the extensive genetic impurity of plants, and the impossibility of inclusion in systematic works of the hundreds of lines which geneticists may be able to segregate from impure species. No doubt, however, many geographical or ecological races of plants are mere expressions of the same principle stipulated above, *i.e.* the segregation and preservation of favoured races. Such races it is convenient and important to recognise taxonomically as subspecies or varieties, whereas the unselected races, occurring together or indiscriminately under identical conditions, are preferably not separately recognised by the systematist but

included in his range of variability for the species. These latter indiscriminate variations also occur among parasites, as has been pointed out, for instance, by Kellogg (1913) for Mallophaga, by Taliaferro (1922) for trypanosomes, and by Dobell and Jepps (1918), and others, for *Entamoeba*. Probably the three, four and five flagellated *Trichomonas* are of similar significance. On the other hand, the selected "hostal" or environmental races of parasites are comparable, to a considerable extent, with the geographical or ecological races of free-living animals, the most essential difference in the comparison being the fact that a parasite living in different hosts has no middle ground where environmental conditions are transitional, and consequently would not be expected to show the intergrading forms which commonly occur between different races of a free-living species. If it were not for the interrupted nature of the spread of a parasitic species to a wider range of hosts, it is very probable that most parasites would be less closely confined to certain hosts than is usually the case. It has been found convenient in the taxonomy of free-living animals to adopt a trinomial system of nomenclature, recognising geographical races or varieties by a subspecific or varietal name following the species name. Although this sytem has been applied in a few instances in parasitology its use has hitherto been very limited, although its usefulness in this field of taxonomy appears to be even greater than in the nomenclature of free-living animals. In view of Nuttall's work on the relationships of the head and body louse, few will question the usefulness and convenience of Nuttall's names *Pediculus humanus capitis* and *P. h. corporis* for these two kinds of lice. In the case of the itch mites, those who do not consider the various forms as distinct species usually refer to the human form as *Sarcoptes scabiei*, and to the forms from animals as *S. s.* var. *equi*, *S. s.* var. *suis*, etc., although it seems to the writer that it would be preferable to retain the name *S. scabiei* for the entire group of related races, designating the typical, *i.e.* first described form, as *S. scabiei scabiei* and the others as *S. s. equi*, *S. s. suis*, etc., omitting the unnecessary and cumbersome "var." preceding the varietal name. The trinomial system of nomenclature lends itself very readily to a large number of instances of closely related parasites, about the specific distinctness of which there is some dispute. One finds the dwarf tapeworms of rats referred to by different writers as *Hymenolepis nana*, *H. murina* (an invalid name), or by the trinomial suggested by Stiles, *H. nana fraterna*. Inasmuch as there are observable average morphological differences between the dwarf tapeworms of man and rats, and there is evidence that the two forms are not *readily* interchangeable between the two hosts, some means of distinction is desirable. Yet to give these two closely related forms the same nomenclatural distinction as is given *H. nana* and *H. diminuta* decidedly grates on one's sense of justness and fitness. As with the itch mites, a better solution would be to refer to both human and rodent forms collectively as *H. nana*, to the human form as *H. nana nana*, and to the rodent form as *H. nana fraterna*. One would find it more convenient and probably more scientifically correct, as indicating re-

lationships, to speak of *Trichomonas hominis* as including both the closely related forms living in the intestine, mouth and vagina of human beings, and the almost identical forms living in the intestine of rodents and other mammals, and to indicate the various forms as *Trichomonas hominis intestinalis*, *T. h. vaginalis*, *T. h. muris*, etc. The same end is commonly reached under our present binomial system by referring to the closely related species collectively merely as *Trichomonas*, which is obviously inaccurate. It may be suggested that this argument might be carried to an absurd point, and if we are to have trinomial names, similar arguments could be brought out in some instances for the use of quadrinomial and quintanomial names. One must, however, strike a happy medium of accuracy and simplicity. In the use of the binomial system too much accuracy is sacrificed for simplicity, whereas in a quadrinomial system too much simplicity would be sacrificed for accuracy. The great success and growing favour with which the trinomial system has met in the nomenclature of free-living animals, particularly birds and mammals, is fairly good evidence that more is gained than lost in the addition of a subspecific or varietal name, and there seems to be much in favour, and little against, its wide application in parasitology.

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A NEW GENUS OF NEMATODES FROM THE DOMESTIC RABBIT.

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(With Plate XI.)

In rabbits maintained for experimental purposes at this Department, there has been collected from the stomach on a number of occasions a nematode showing certain affinities with the genus *Graphidium* Railliet and Henry, 1909. The parasite in question, however, differs from the type species of that genus in characters which must be regarded of more than specific value and it has therefore been assigned to a new genus. It has only recently been noticed in our rabbits. I am indebted to Dr Smith for calling my attention to it. Whether the parasite had heretofore been overlooked in autopsy examinations or has been recently introduced cannot be said. It seems probable that it originated from a wild host and is indigenous to this continent. It was first noticed after our rabbits had been transferred to quarters where they had access to the ground.

The parasite falls in the family Trichostrongylidae Railliet, 1915, and the subfamily Trichostrongylinae Leiper, 1908. The name proposed for it is *Obeliscus cuniculi*.

Generic diagnosis. Obeliscus. Head nude. Cuticle marked by a limited number of prominent longitudinal striae. Cervical and prebursal papillae present. Bursa deeply incised to form two lateral lobes. A small, distinct, well defined dorsal lobe, supported by a dorsal ray. Rays of lateral lobe fall into the usual ventral, lateral and dorsal groups. The ventro-ventral and latero-ventral rays after a pronounced divergence terminate near each other. The medio- and postero-lateral rays originate in the same stalk and are parallel. The spicules are cleft distally, the branches terminated by a recurved hook. Vulva toward posterior end of body. Well developed muscular ovijectors are present.

Description of the Species.

The worms are whitish in colour but show some dark streaking due to the colour of the intestine. The anterior end is blunt. The cuticle is very finely striated transversely and marked by prominent straight or undulating longitudinal striae numbering approximately 27 to 36. Two short, stout,

backwardly directed cervical papillae are present in the form of bluntly pointed spines. There is no pharynx. The oesophagus after a very slight initial reduction in diameter gradually expands to the broad posterior end where it is rounded off (Pl. XI, fig. 1).

The male. The length ranges from 10 to 14 mm. The maximum width occurs toward or at the posterior end. It was 211 and 229 μ in two instances. The body tapers gradually to the anterior end. The head measured 57 μ broad. The cervical papillae were located 581 μ from the anterior end. The number of longitudinal striae of the cuticula as determined near the middle of the body is approximately 27. There are a pair of prebursal papillae. The measurement of the oesophagus in two instances was 810 and 827 μ long and 103 and 90 μ in maximum breadth respectively. The spicules are brown in colour, nearly straight (Fig. 2), equal in length, and in two males measured 440 and 475 μ long and 27 μ broad. Very close to the proximal end they are provided with a thin, rounded, concave expansion projecting at an angle to the axis of the spicule, and at the distal end they are cleft for a short distance. One branch is flattened and enfolds the other laterally and ventrally ending in an inwardly directed recurved hook from the summit of which projects a relatively broad process rounded at the end. The other branch ends in a recurved hook directed dorsally, from the crest of which projects a very fine, curved spine (Fig. 3). The bursa (Fig. 4) consists of two large, rounded, lateral lobes and a small dorsal lobe. The internal surface of the former, except at the periphery, is thickly covered with small round papillae variable in size. Peripherally the same surface is marked by radiating ridges which may show branching. Each lateral lobe is supported by six well-developed rays. The ventro-ventral ray is small and bends slightly ventral. It originates from the base of the latero-ventral ray. The latter is large and after passing backward for more than half its length curves ventral and terminates near the end of the former. The externo-, medio-, and postero-lateral rays form the usual lateral system. The former is large and runs along the middle of the lobe terminating near its margin. The other two are small, lie close together, and bend slightly dorsal. The externo-dorsal ray is small and slightly curved.

The dorsal lobe of the bursa (Fig. 5) is median, distinctly demarked from and lies within the lateral lobes. It may be looked on as slightly three-lobed, there being two lateral and a very small terminal median lobe. The end of the dorsal ray extends into the latter where it divides into two branches, each of which bifurcates, the external portions being the shorter. A little anterior to the first division two lateral spurs are given off (Fig. 4).

The female. It ranges in length from 15 to 18·5 mm. The maximum width (387 and 546 μ in two instances) occurs at the middle of the body. The width of the head is 119 μ. The tail is pointed. From the middle region the body gradually tapers in both directions but a few millimetres (3·2 to 3·6) from the posterior end there is an abrupt reduction in diameter (Fig. 6). In a specimen measured this region had an anterior and middle diameter of 264 and 282 μ

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In *Parasitology*, vol. xv, p. 340 (20. ix. 1923), I described a genus of nematodes from the rabbit for which I proposed the name *Obeliscus*. Dr. Paul Bartsch, of the Smithsonian Institution, has called my attention to the fact that this name is preoccupied, having previously been used for genera of Mollusca by Humphreys in 1797, by Agassiz in 1837, and by Beck in 1837. I therefore propose the new name *Obeliscoides*, type *cuniculi*, to replace *Obeliscus* Graybill, 1923.

ADDENDUM.

The accompanying slip is arranged to be pasted below the text on page 586 of Vol. xlviii of the *Studies*.

respectively. The vulva is a transverse slit located at the anterior end of this region. In two measurements made the anus was located 226 μ and 299 μ from the posterior end. The cervical papillae were located 649 μ from the anterior end. The longitudinal striae of the cuticula number approximately 32 to 36. The oesophagus measured 1 mm. long. The diameter near the anterior end was 37 μ and the posterior diameter in two specimens 131 μ and 144 μ. The nerve ring occurred 405 μ from the anterior end. The vagina is short leading directly inward into well-developed muscular ovijectors (Fig. 6). The terminal portions of the uteri enter the ovijectors anteriorly and posteriorly. This region of the uterus is separated from the rest by a constriction, is somewhat narrower and appears to be specialised for passing the ova into the ovijector.

The ova are ellipsoidal in shape (Fig. 7). They are provided with a very thin, double-contoured shell. When deposited they are in the morula stage. In size they range from 76 to 86 μ long and 44 to 45 μ broad.

This species shows certain relations to *Graphidium strigosum* (Duj. 1845) Railliet and Henry, 1919, type species of *Graphidium*, reported from Europe in rabbits (*Oryctolagus cuniculus* and *Lepus europaeus*). The more striking structural differences of the latter are as follows(1):—The spicules are long, filiform, and frayed out at the distal end. There is no dorsal lobe to the bursa. The five rays of the lateral and ventral systems are uniformly and symmetrically spaced, and the externo-dorsal ray is long and prominent. These differences are considered sufficiently important to warrant placing the form here described in a new genus.

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EXPLANATION OF PLATE XI.

All drawings except Figs. 3 and 5 were made with a camera lucida.

Fig. 1. Anterior end of a male, showing oesophagus and cervical papillae. ×103.
Fig. 2. Spicules. ×170.
Fig. 3. End of a spicule showing the two branches.
Fig. 4. Dorsal view of bursa. Shape of dorsal lobe distorted. ×80.
Fig. 5. Outline of dorsal lobe of bursa showing dorsal ray.
Fig. 6. Body of female in region of vulva showing beginning of narrow posterior portion of the body, muscular ovijectors, uteri, ovaries, and intestine. ×103.
Fig. 7. Drawing of ovum when deposited. ×680.

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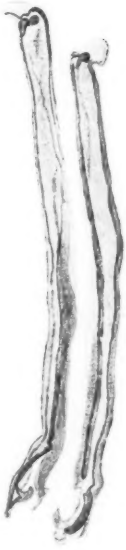

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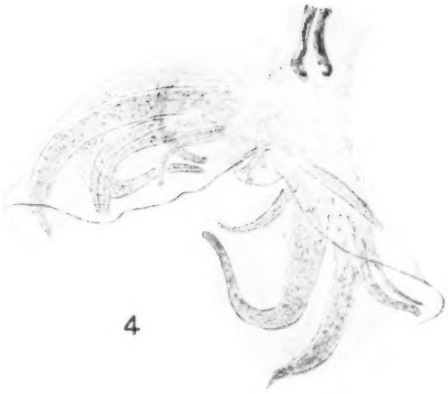

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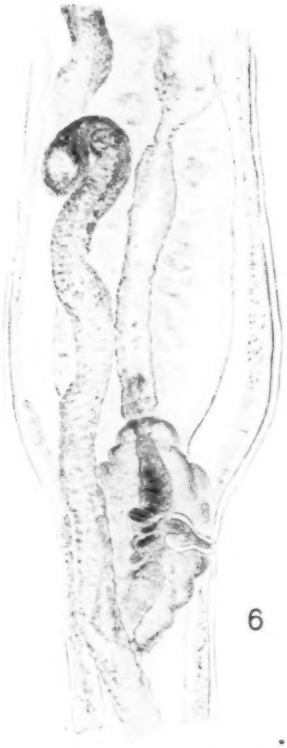

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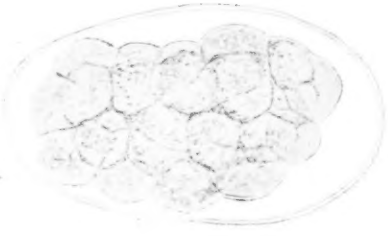

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